A COMPARISON BETWEEN THREE DIFFERENT DOSES OF INTRATHECAL DEXMEDETOMIDINE ADDED TO HYPERBARIC BUPIVACAINE FOR INFRA UMBILICAL SURGERIES

A STUDY OF 60 CASES

DISSERTATION

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BRANCH X -ANAESTHESIOLOGY

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CERTIFICATE

This is to certify that this dissertation "**A COMPARISON BETWEEN THREE DIFFERENT DOSES OF INTRATHECAL DEXMEDETOMIDINE ADDED TO HYPERBARIC BUPIVACAINE FOR INFRA UMBILICAL SURGERIES**" presented herein by**Dr.K.PREMAKUMARI** is an original work done in the Department of Anaesthesiology, Tirunelveli Medical College Hospital, Tirunelveli for the award of Degree of M.D (Branch - X) Anaesthesiology under my direct supervision and guidance, during the academic period of 2010 – 2013.

DEAN

Tirunelveli Medical College

Tirunelveli-627007

CERTIFICATE

This is to certify that the Dissertation "**A Comparison Between Three Different Doses Of Intrathecal Dexmedetomidine Added To Hyperbaric Bupivacaine For Infra Umbilical Surgeries**" presented herein by **Dr. K. PREMAKUMARI** is an original work done in the Department of Anaesthesiology, Tirunelveli Medical College Hospital, Tirunelveli for the award of degree of M.D (BranchX) Anaesthesiology under my guidance and supervision during the period of 2010 - 2013.

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DECLARATION

I,Dr.K.PREMAKUMARI declare that the dissertation titled "**A comparison between three different doses of intrathecal dexmedetomidine added to hyper baric bupivacaine for infra umbilical surgeries"** has been prepared by me.

This is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.D. degree, Branch X (ANAESTHESIOLOGY) Degree Examination to be held in April 2013.

Place: Tirunelveli DR.K.PREMAKUMARI

Date:

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INTRODUCTION

"It is the duty of the anesthesiologist to study the well-being o fthe patient as well as the convenience of the surgeon"

-R.M.WATERS

"*It is not the drug that is dangerous, but the man who administers it is"*

- SIR ROBERT MACINTOSH

The term ''spinal anesthesia''was coined in 1885 by Leonard Corning, a neurologist. First planned spinal analgesia for surgery in man was performed by August Karl Gustav Bier on 16th August 1898 in Kiel and he was credited for introducing spinal anesthesia.Heinrich Quincke of Keil, Germany described the technique of lumbar puncture.

Spinal anesthesia using local anesthesia is associated with relatively short duration of action and hence early analgesic intervention is needed in post operative period.A common problem during infra umbilical surgery under spinal anesthesia is visceral pain, nausea and vomiting.

Adjuvants are added to improve the quality, to accelerate the onset of action and also to overcome the problems which occur during spinal analgesia.Adrenaline was the first spinal adjuvant used. Adrenaline reduces its toxicity but does not greatly prolong its effect.

Various adjuvants like morphine, fentanyl, sufentanil, clonidine, midazolam, ketamine, neostigmine, sodabicarbonateare added to local

anesthetics and the latest inclusion is dexmedetomidine[6]. Adjuvants are administered by various routes like epidural, intrathecal and intravenous. In our study adjuvant is added to local anesthesic through intrathecal route.

Alpha 2 adrenergic receptor agonist like dexmedetomidine gain the focus of interest for its sedative, analgesic, perioperative sympatholytic and hemodynamic stabilizing properties. Dexmedetomidine is a new highly selective drug among the alpha 2 adrenergic receptor agonist. It has been approved by FOOD AND DRUGS ADMINISTRATION for short term sedation for mechanically ventilated ICU patients. No neurological defects have been reported till date in both human and animal studies during intrathecal use. This study is intended to compare three different doses of intrathecal dexmedetomidine added to hyperbaric bupivacaine for infra umbilical surgeries (Unilateral Inguinal Hernia surgeries and Vaginal Hysterectomies)

AIM AND OBJECTIVES

To compare the effects of 3 different doses of intrathecal dexmedetomidine added to hyper baric bupivacaine for infraumbilical surgeries(Unilateral inguinal hernia and Vaginal Hysterectomies) with respect to

1. Onset of sensory and motor blockade
2. Duration of sensory and motor blockade
3. Hemodynamic effects
4. Duration of post operative analgesia
5. Post operative sedation

HISTORY OF PAIN

Pain – It is derived from a Greek word ‘’poine’’ which means penalty. CHARLES BELL and FRANCOIS MAGENDIE demonstrated that dorsal roots of the spinal cord transmit sensory information whereas ventral roots transmit motor information and the idea of specific neural pathway for painful sensations originated.

Alpha 2 agonist have been used by veterinarians for many years for regional analgesia, but is being used in humans since 12 years.

PHYSIOLOGY OF PAIN

Pain is a complex phenomenon which includes sensory – discriminative and motivational – affective components. The sensory component depends on ascending projection of tracts like spinothalamic and trigeminothalamic tracts into the cerebral cortex. They perceive quality of pain and help to know location of stimulus, intensity and duration of stimulus.

Affective component includes attention, arousal, somatic, autonomic reflex, endocrine and emotional changes

Pain receptors (Nociceptors):

Nociceptors are free nerve endings seen in the skin, muscles, viscera, joints and vasculature. Nociceptors detect the noxious stimulus due to the chemical, mechanical and thermal (heat & cold) changes. They can be classified into exteroceptors, which receive stimuli from skin surface and interoceptors that are located in the walls of viscera or deeper structures. These are free nerve terminals and are seen adjacent to small blood vessels and mast cells. Nociceptors operate as a functional unit with these.

In addition to nociceptors, somatosensory receptors are located in the skin, which are sensitive to other forms of stimulation and each sensory unit includes an end-organ receptor, accompanying axon, dorsal root ganglion, and axon terminals in the spinal cord.

Inthe gate control theory of pain, Melzack and Wall (1965) proposed that inhibitory interneurons located in the superficial part of the dorsal horn played a crucial role in controlling incoming sensory information before it was transmitted to the brain through ascending pathways.

The dorsal horn contains four major neuronal components:

1. The central terminals of primary afferent axons
2. Intrinsic neurons
3. Projection neurons
4. Descending axons that pass caudally from several brain regions.

THE LAMINA OF REXED

Rexed (1952) divided the dorsal horn of the cat spinal cord into a series of six parallel laminae, based on differences in the size and packing density of neurons (cyto architectonics). Lamina II can be subdividedinto two parts, referred to as lamina II inner (IIi) and lamina II outer (IIo). Laminae I and II are referred to as the superficial dorsal horn, constitute the main target for nociceptive primary afferents. The deeper laminae (III - VI) also have an important role in pain. Some nociceptive primary afferents terminate in this region, and many of the neurons in these laminae including some projection cells are activated by noxious stimulation.

Lamina I, also called as marginal layer, forms a thin two dimensional sheet covering the dorsal aspect of the dorsal horn and contains both projection neurons and inter neurons.

A few large projection neurons are called as giant marginal cells of Waldeyer. Lamina II is also known as the substantia galatinosa, because the lack of myelinated fibres within it gives it a translucent appearance in unstained sections. Lamina III also contains a high density of neurons. Laminae IV - VI are more heterogeneous, with neurons of various sizes, some of which are projection cells.

PRIMARY AFFERENT FIBRES:

The somata of primary sensory neurons that innervate the limbs and trunk are located in sensory gangliaassociated with spinal nerves (dorsal root ganglia). Their axons bifurcate within the ganglion giving rise to peripheral and central branch, where it forms synapses with second-order neurons. Fibres innervating skin are described as cutaneous sensory neurons.

Afferent fibres innervating abdominal or pelvic viscera are termed visceral afferents.

Cutaneous sensory neurons:

- Myelinated low-threshold mechanoreceptors
- Myelinated nociceptive afferent fibres
- Unmyelinated afferent fibres

Receptors associated with primary afferent neurons:

Primary afferent fibres also possess a rich diversity of ligand-gated ionotropic, metabotropic and tyrosine kinase receptors which include both the alpha - amino - 3 - hydroxy - 5 - methyl - 4 - isoxazolepropionic acid (AMPA) and N - methyl - D - aspartate (NMDA) classes of ionotropic glutamate receptors and metabotropic glutamate receptors.

Lastly, α_2 adrenergic receptors are also found in sensory neurons and are thought to be localised at the central terminals of peptidergic fibres[1]

PROJECTION NEURONS, SUBSTANCE P AND THE NEUROKININ 1 RECEPTOR:

Neurons with axons that project to the brain are present in large numbers in lamina I and are scattered through the deeper part of the dorsal horn (laminae III - VI) and the ventral horn.

Lamina I and some of the projection cells in deeper laminae, have axons that cross the midline and ascend to a variety of supra spinal targets including the thalamus, the midbrain periaqueductal grey matter, lateral para-brachial area of the pons and various parts of the medullary reticular formation.

Substance P is present in many nociceptive primary afferents[2] and there is evidence that this peptide and the neurokinin I (NKI) receptor, on which it acts, have a significant role in spinal pain mechanisms[3].

Substance P is released from primary afferents at extra synaptic sites and acts on NKI receptors on the projection neurons through volume transmission.

SPINAL INTERNEURONS:

Interneurons make up the great majority of the neuronal population throughout the dorsal horn, laminae I - III contains a large number of interneurons since the packing density of neurons is particularly high.

Classification of interneurons inhibitory and excitatory interneurons:

Inhibitory interneurons can be subdivided into those that use GABA but not glycine as transmitters and that use both.

Most excitatory interneurons are glutamatergic.

GABA and glycine receptors:

$GABA_A$ and glycine receptors are widely distributed in the spinal cord and are probably expressed by all dorsal horn neurons.

DESCENDING MONOAMINERGIC AXONS:

Serotoninergic axons in the spinal cord originate in the medullary raphe nuclei, while those that contain norepinephrine are derived from cells in the locus ceruleus and adjacent areas of the pons.

Serotonin containing axons are widely distributed throughout the dorsal horn, but are numerous in laminae I and IIo.

Norepinephrine containing axons can be identified with antibodies against appropriate synthetic enzymes (eg. dopamine - β hydroxylase). They are found throughout the dorsal horn, with high density in laminae I and II[4].

ANATOMY OF SUB ARACHNOID SPACE

The spine consists of 33 vertebrae (seven cervical, twelve thoracic, five lumbar,five fused sacral, four fused coccygeal).With the exception of C1 the cervical, thoracic and lumbar vertebrae consist of a body anteriorly, two pedicles that project posteriorly from the body and two laminae that connect the pedicles. These structures form the vertebral canal which contains a spinal cord, spinal nerves and epidural space.

The laminae give rise to the transverse processes that project laterally and the spinous process that projects posteriorly. These bony projections serve as sites for muscle and ligament attachments. The pedicles contain a superior and inferior vertebral notch through which the spinal nerves exit the vertebral canal.

The first cervical vertebrae differs from the typical structure in that it does not have a body or a spinous process. The five sacral vertebrae are fused together to form the wedge shaped sacrum which connects the spine with the iliac wings of the pelvis. The fifth sacral vertebrae is not fused posteriorly, giving rise to sacral hiatus. The sacral cornu are bony prominences on either side of the hiatus. The four rudimentary coccygeal vertebrae are fused together to form the coccyx. A line drawn between the iliac crests crosses the body of L5 or L4 -L5 interspace.

LIGAMENTS

The vertebral bodies are stabilized by five ligaments that increase in size between the cervical and lumbar vertebrae.

- Supraspinous ligament
- Ligamentum nuchae
- Ligamentum flavum
- Anterior longitudinal ligament
- Posterior longitudinal ligament

Ligamentum flavum is a tough wedge shaped ligament composed of elastin. It consists of right and left portions that span adjacent vertebral laminae and fuse in midline to varying degrees. The ligamentum flavum is thickest in the midline, measuring 3-5 mm at the L2-3 interspace of adults.

SPINAL MENINGES

These spinal meninges consist of three protective membranes (duramater, arachnoid mater and piamater) which are continuous with the cranial meninges.

Duramater

The duramater is the outermost and the thickest meningeal tissue. The spinal duramater begins at the foramen magnum where it fuses with periosteum of the skull, forming the cephalad border of the epidural space. Caudally, the duramater ends at approximately S2 where it fuses

with the filumterminale. It is composed of collagen fibres and elastin fibres arranged longitudinally and circumferentially. The inner surface of the duramater abuts the arachnoid mater. There is a potential space between these two membranes called the subdural space. The incidence of subdural injection during intended subarachnoid injection may be as high as ten percentage as per radiological literature[5].

Arachnoid mater

The arachnoid mater is a delicate, avascular membrane composed of overlapping layers of flattened cells with connective tissue fibers running between the cellular layers. The arachnoid mater herniates through the duramater into epidural space to form arachnoid granulations. The subarachnoid space lies between the arachnoid mater and piamater and contains the cerebro spinal fluid. Spinal cerebro spinal fluid is in continuity with the cranial cerebro spinal fluid and provides a venue for drugs in the cerebrospinal fluid to reach the brain. The spinal nerve roots and rootlets run in the subarachnoid space.

Piamater

The spinal piamater is adherent to the spinal cord and is composed of a thin layer of connective tissue cells interspersed with the collagen. The piamater extends to the tip of the spinal cord where it becomes the filumterminale which anchors the spinal cord to the sacrum.

SPINAL CORD

In the first trimester fetus,the spinal cord extends from the foramen magnum to the end of the spinal column.Thereafter the vertebral column lengthens more than the spinal cord so that at birth the spinalcord ends at about the level of the third lumbar vertebra.In the adult,the caudad tip of the spinalcord lies at the level of first lumbar vertebrae.The adult spinal cord measures approximately 41-48cm in length and weighs between 24-36g,about 1 cm in diameter.The tapered end of the cord is called the conusmedullaris.The spinal cord gives rise to 31 pairs of spinal nerves,each composed of an anterior motor root and a posterior sensory root.

PHYSIOLOGY OF SUB ARACHNOID BLOCK

The cerebrospinal fluid is an ultrafiltrate of the blood plasma, it is a colourless, clear fluid, present in spinal and cranial sub arachnoid space and in the ventricles of the brain. Average volume of CSF in adult is 120-150 ml, among this 35 ml seen in the ventricles and 25 ml in the cerebral sub arachnoid space and 75 ml in the spinal sub arachnoid space. CSF is secreted by choroid plexus at a rate of about 0.3-0.4 ml/minute.

PHYSICAL CHARACTERISTICS OF CSF

The pH is 7.4. The specific gravity is 1.007 and density is 1.0003, baricity is 1.000and the CSF pressure varies between 8-12 mm Hg, cell count is 3-5/ cumm and the protein content is 20 mg/dl and glucose content varies between 40-80mg /dl.

The CSF mainly plays a role in spinal anesthesia and it serves as a media for dispersionof the local anaesthetic drug in the spinal nerve

Spread of the drug in sub arachnoid space is determined by the specific gravity of the injected drug when compared with that of CSF.

MECHANISM OF SPINAL ANESTHESIA

The nerves of the sub arachnoid space do not have the protection of dura or arachnoid, therefore even a small amount of local anaesthetic in the CSF will cause a profound block of nerve transmission.

Local aesthetics for spinal anesthesia is usually injected into the sub arachnoid space between the spinous processes of the third and fourth

lumbar vertebra, the needle will enter the Dura in the area of the caudaequina, the place where the nerve roots cross the sub arachnoid space from the spinal cord to the point of exit through the dura. Surface area of nerve roots is considerable, thus making them vulnerable to the effects of local anaesthetic. Local anaesthetics penetrate the smaller roots more rapidly because of the largest surface area[7]. Spinal cord also takes up local anaesthetic mainly by diffusion through piamater. But the concentrations of local anaesthetics are higher in nerve roots than in the cord because of easy accessibility of local anaesthetic. Local anaesthetics cause sodium channel blockade within the dorsal and ventral horns, thus inhibits the generation and propagation of electrical activity

The block and recovery of sensory fibres occurs in this order: The most sensitive sensory fibres-C fibres (sensation to cold)-are blocked first and remain blocked longest; A delta fibres (pin-prick)are the second to be blocked and recover; A β fibres(touch)are the last to block and first to recover.

The preganglionic sympathetic fibres (B –fibres) are most sensitive to local anaesthetics. The motor fibres (Aα, the largest fibers) are less sensitive to local anaesthetics comparing to sensoryfibers and there is a difference between sensory and motor block, motor function is better preserved since more local anaesthetic is needed to anesthetize the thick motorfibers.

UPTAKE AND ELIMINATION OF LOCAL ANESTHETICS FROM CEREBROSPINAL FLUID

Factors affecting uptake of local anaesthetic (LA) into neural tissue:

- Concentration of LA in cerebrospinal fluid(CSF)
- Surface area of tissue exposed to CSF
- Lipid content of nerve
- Blood flow of nerve

Elimination of LA from CSF:

- Through the arachnoidea and dura to epidural space
- Vascular absorption via sub arachnoid and epidural blood vessels

Spread of Local Anaesthetics in Sub arachnoid Space

The main factor influencingthe spread of drug in the CSF is the relationship between density of the local Anaesthetic in relation to the density of the CSF at a specific temperature called baricity. Density of a solution is the weight in grams /ml of a solution(g/ml)at a specified temperature. Anaesthetic substance that have a greater density than CSF are called hyperbaric and those with the lower density are called hypobaric and local anaesthetic with density close to the CSF are called isobaric and factors like gender and hormonal status in women(menopause, pregnancy)affect on CSF density. With hyperbaric solutions the spread is more influenced by baricity and the duration of

block also increases when the dose is increased when hyperbaric solutions are used[8,9]. Hypobaric and hyperbaric solutionsare prepared by adding distilled water and dextrose to isobaric solution respectively. Gravity does not influence the spread of isobaric solution and hence height of block is not influenced by changing the position of the patient. Hyperbaric solutions settle to the most dependent aspect of the sub arachnoid space and since it is heavier than CSF in supine position, hyperbaric solution spread to the level of thoracic kyphosis and hypobaric solution floats up.

INDICATION

Infra Umbilical surgeries, lower limb surgeries and urological surgeries, obstetric and gynaecological surgeries and surgeries around the perineum. Spinal anesthesia can be combined with epidural anesthesia for anesthesia in obstetrics, vascular and orthopaedic surgeries.

CONTRAINDICATIONS

Absolute Contraindications

- Patients refusal despite adequate information.
- Infections at the site of injection
- Dermatologic conditions
- Septicemia or Bacteremia
- Shock or severe hypovolemia

- Abnormality in blood clotting mechanism
- Increased intracranial pressure
- Lack of skill in spinal anesthesia
- Allergy to local anesthesia

Relative contraindications

- Deformities of the spinal column
- Pre-existing disease of the spinal cord
- Chronic head ache or back ache
- Inability to achieve a spinal tap in three attempts
- Cardiac diseases – marked aortic stenosis

SPINAL ANESTHESIA TECHNIQUE

PREPARATION AND MONITORING OF THE PATIENT

Proper patient selection is mandatory for successful procedure, which includes thorough pre anesthesic evaluation Wide bore IV line, blood pressure and heart rate monitor, pulseoximeter, equipment for airway management, working suction apparatus, two oxygen cylinder and emergency drugs(atropine, ephedrine) kept ready in the syringes.

POSITION OF THE PATIENT

Lateral decubitus position is the most popular position because of comfort. The patient should be placed with the back parallel to the edge of the table closest to the anesthesiologist. The vertebral column is then flexed to widen the interlaminar spaces, by drawing the knees upto the

chest and putting the chin down on the chest, the head supported by a pillow.

Sitting position is used for obstetrical, certain gynaecologic, and urologic procedures. This position facilitates identification of the midline particularly in obese patients in whom there will be difficulty

NEEDLES FOR SPINAL ANESTHESIA:

Needles either of small bore or with a rounded, non-cutting bevel are used.

The Quincke-Babcock spinal needle (needle with sharp point with a medium length cutting bevel), the Whitacre needle and the Sprotte needle (needles of completely rounded, non cutting bevels with solid tips, openings are on the side, 2 to 4 mm proximal to the tip) are used.

Quincke type:

Opening at the tip causing injectate to flow in a straight direction.

Sprotte type:

As hole in the side, the flow is directed approximately 45 degrees from the longitudinal axis.

ASEPTIC TECHNIQUE

Before the spinal anesthesia, the anesthesiologist must perform a thorough surgical scrub using alcohol-based antiseptic solutions.

The patient's back is prepared with alcohol based antiseptic solution and sterile drapes are applied. The insertion site for lumbar puncture should

be identified by the line between the upper border of the iliac crests, which passes through either the spinous process of L4 or the interspace between L4 and L5. Spinal needle is introduced through Midline approach either in sitting /right lateral decubitus position.26 gauge spinal needle is used and the needle is introduced through middle of the interspace and after piercing the skin and subcutaneous tissue , it is advanced in a cephalad direction with the long axis of the vertebral column. Stylet is gently removed, appearance of CSF through the hub of the needle confirms correct position of needle, and the stylet is again inserted to prevent leakage of CSF.The hub of the needle is held between thumb and index finger of the anesthesiologists non dominant hand and syringe is attached to the needle, gentle aspiration done to confirm free flow of spinal fluid and the drug is injected. Then the patient is placed in supine position continuous monitoring of vital parameters done and level of analgesia confirmed byloss of sensation to pinprick. Motor block was assessed by modified Bromage score.

PHYSIOLOGIC RESPONSES

A: EFFECTS ON CARDIOVASCULAR SYSTEM[10]

The responses are due to combined effects of autonomic denervation, with higher levels of blockade, the added effects of vagal nerve innervation. Spinal anesthesia causes some degree of hypotension

and reflex bradycardia because of reduction in cardiac output and systemic vascular resistance.

The level of sympathetic denervation determines the magnitude of cardio vascular responses to spinal anesthesia, the higher the level of neural blockade, the greater the change in cardio circulatory parameters. Sympathetic denervation produces arterial and arteriolar vasodilation which is not maximal, whereas veins and venulesvasodilate maximally due to loss of vascular smooth muscle tone.

The bradycardia seen during spinal anesthesia is due to blockade of preganglionic cardiac accelerator fibres arising from T_1 to T_4 during high levels of anesthesia[11].

The bradycardia is also mediated by significant decreases in right atrial pressure and pressure in the great veins as they enter the right atrium. The direct relationship between right atrial pressure and heart rate during high spinal anesthesia is mediated by intrinsic chronotropic stretch receptors located in the right atrium and adjacent great veins, the mechanism for these changes is described as Bezold-Jarisch reflex.

EFFECTS ON RESPIRATORY SYSTEM

High spinal anesthesia cause intercostal paralysis, Arterial blood gas tension, resting tidal volume, maximum insipiratory volume, remain unaltered because diaphragmatic activity is unimpaired. Maximum

breathing capacity and maximum expiratory volume are diminished. Phrenic nerves are unaltered.

Gastro Intestinal Effect:

Preganglionic fibres from T5-L1 are inhibitory to gut. So in sympathetic blockade the small intestine contracts with relaxed sphincters and peristalsis remains normal. Handling of viscera causes discomfort and bradycardia since vagus is not blocked.

Hepatic and Renal Effects:

The hepatic blood flow decreases and is directly proportional to thedecrease in blood pressure. There may be normal hepatic oxygenextraction. Renal blood flow is maintained by autoregulation and does not decrease till mean arterial pressure goes below 50mmHg.

Genito Urinary System:

Sphincters of bladder are not relaxed, and the ureteric tone are not greatly altered. Urinary retention occurs. Penis is often engorged. Uterine tone is unchanged in pregnancy. In the absence of hypotension spinal anesthesia has got no effect on the progress of labour and uterine blood flow.

Metabolic and hormonal effect:

Spinal anesthesia blocks the hormonal and metabolic responses to nociceptive stimuli arising from the operative site. It minimizes the rise in blood sugar, cortisol, catecholamines, renin and aldosterone release

associated with stress. Post operative negative nitrogen balance and secretion of antidiuretic hormone are inhibited

THERMO REGULATION

Extensive spinal blockade impairs central thermo regulatory control[12]. The main cause of hypothermia during spinal anesthesia is the redistribution of blood flow and heat to the periphery because of vasodilation.

COMPLICATIONS OF SUB ARACHNOID BLOCK

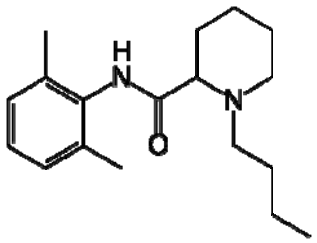
Immediate

- Hypotension
- Bradycardia
- Toxicity due to intravascular injection
- Allergy to local anaesthetic drug.
- Hypotension(brainstem hypoxia)

Late

- Post dural puncture headache
- Retention of urine
- Backache
- Meningitis
- Transient lesions of caudaequina
- Sixth nerve palsy
- Anterior spinal artery syndrome

PHARMACOLOGY OF BUPIVACAINE:



Bupivacaine is an amide type local analgesic drug. It is a hydrochloride salt of 1-butyl-N-(2, 6-dimethylphenyl) piperidine-2-carboxamide.

It was synthesized in Sweden by Ekenstam and his colleagues in 1957.

First used clinically by L.J. Telivuo in 1963.

Pka is 8.2

Molecular weight	-	288
Protein binding	-	95%
Lipid solubility	-	28
Elimination half life	-	210mts
Toxic plasma concentration	-	>1.5µg/ml
Approximate duration of action	-	175mts

The drug is very stable to acids, alkalis and repeated autoclaving. Bupivacaine 0.5% is the preferred strength. Higher concentration result in greater variability of spread[13]. Bupivacaine is 4 times as potent as lignocaine, hence 0.5 % solution is approximately equivalent to 2 %

lignocine. It is more cardiotoxic than lignocaine and which is aggravated by hypoxia, hypercapnia and by pregnancy. It causes more sensory than motor block .It is not recommended for intravenous regional analgesia. Duration of effect is between 5 and 16 hours and is one of the longest acting local analgesics, which is related to binding to nerve tissue. Small percentage of a given dose of drug is excreted unchanged in the urine and the remainder is metabolised in the liver.

Uses:

- Spinal anesthesia
- Epidural anesthesia
- Caudal anesthesia
- Continuous epidural anesthesia
- Peripheral nerve block
- Infiltration anesthesia

Onset time and duration of action

Site of action	***Onset (minutes)***	***Duration (minutes)***
Intrathecal	5	90-120
Epidural	15-20	165-225
Brachial plexus	10-20	600

Pharmacokinetics:

Once injected intrathecally, it gets absorbed by the nerve rootlets and it is rapidly absorbed from the site of injection, but the rate of absorption depends on the vascularity and the presence of vasoconstrictors.

Because of high lipid solubility it easily penetrates nerve and vascular tissue. 80-95% of absorbed bupivacaine binds to the plasma proteins.

Distribution:

Rapid distribution phase: (α)

Slow disappearance phase: (β)

Biotransformation:

Possible pathways of metabolism of bupivacaine include aromatic hydroxylation and conjugation. Only the N-dealkylated metabolite, N-desbutyl bupivacaine has been measured in blood (or) urine after epidural (or) spinal anesthesia. Alpha1 acid glycoprotein is the most important plasma protein binding site of bupivacaine and its concentration is increased by many clinical situations including post operative trauma.

Excretion:

It is through the kidney, 4-10% of the drug is excreted unchanged.

Mode of Action:

a) Site of action:

i) The spinal nerve rootlet fine nerve filaments having a large surfacearea are exposed to the local anaesthetics.

ii) Posterior and lateral aspects of the spinal cord.

b) Sodium Channel blockade:

They impede sodium ion access to the axon interior by occluding the transmembrane sodium channels thus delaying the process of depolarization and axon remains polarized. It is a non-depolarisationblockade.

Pharmacodynamics:

It has got a longer duration of action but a slower onset.

Cardiovascular system:

It reduces cardiac output by reducing the sympathetic tone, by slowing the heart rate and by reducing the venous return, it produces a fall in arterial blood pressure but it is relatively slow and is seldom very profound. It produces a fall in central venous pressure. It causes an increase in lower limb blood flow. It causes a reduction in incidence of deep vein thrombosis.

Respiratory System:

It relaxes bronchial smooth muscle. It causes apnea due to phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to drug.

Gastro intestinal tract:

There is an increase in gastro intestinal motility and emptying of the gastric contents are better.

Toxicity:

Toxicity is related to plasma level of unbound drug and more likely due to an inadvertant intravenous injection. Systemic toxicity reactions primarily involve central nervous system and cardio vascular system. The blood level required to produce central nervous system toxicity is less than that required to produce circulatory collapse.

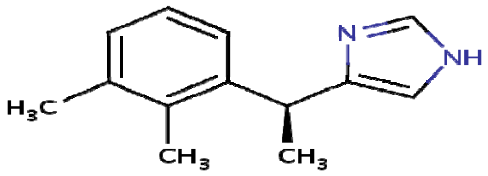
Central Nervous System Toxicity:

Early symptoms are circumoral numbness, tongue paresthesia and dizziness. Sensory complaints include tinnitus and blurred vision. Excitatory signs (restlessness, agitation, nervousness, paranoia) often precede central nervous system depression (slurred speech, drowsiness, unconsciousness). Muscle twitching heralds the onset of tonic clonic seizures. Respiratory arrest often follows. The excitatoryreactions are the result of selective blockade of inhibitory pathways.

Cardiovascular System Toxicity:

The rate of depolarization in fast conducting tissue of purkinje fibres and ventricular muscle is decreased. The rate of recovery of bupivacaine induced block is slower than that of lignocaine.Extremely high concentration of the drug causes sinus bradycardia, hypotension, atrioventricular heart block, idioventricular rhythms, and life threatening arrhythmias such as ventricular tachycardia, ventricular fibrillation and cardiac arrest.

PHARMACOLOGY OF DEXMEDETOMIDINE



Dexmedetomidine is an$\propto_2$- agonist that received FDA approval in 1999. It is used as a short-term sedative analgesic especially in the ICU. It is used less than 24 hrs. Dexmedetomidine is a selective $\propto_2$ – adrenoceptor agonist. It is used in high doses for sedation and analgesic. It is shorter-acting drug than clonidine. and it has a reversal drug Atipamezole for its sedative effect. It is used in perioperative period as sedative and analgesic, as premedication, as an anesthesic adjunct for

general as well as regional anesthesia and also for post operative sedative and analgesic[14].

Physiology of $\propto_2$ -adrenoceptors.

Alpha 2 – adrenoceptors are found in peripheral and central nervous systems, also in effector organs like liver, kidney, pancreas,eye, vascular smooth muscles and platelets.

They are divided into 3 subtypes. $\propto_2$ A- predominant subtypes in CNS, this is responsible for the sedative, analgesic and sympatholytic effect.

Dexmedetomidine is 8 to 10 times more selective towards $\propto_2$ AR than Clonidine. $\propto_2$ B –found mainly in the peripheral vasculature, and is responsible for the short term hypertensive response.

$\propto_2$ C-found in the CNS, Which is responsible for the anxiolyticeffect[15].

All these subtypes produce cellular action by signalling through a G-Protein which couples to effector mechanisms, and the coupling differs depending on receptor sub-type and location. The $\propto_2$ A-Subtype appears to couple in an inhibitory fashion to the calcium channel in the locus ceruleus of the brain stem and in the vasculature, the $\propto_2$ B subtype couple in an excitatory manner to the same effector mechanism.

Mechanism of action of dexmedetomidine:

Dexmedetomidine possess unique properties and it differs from other sedative drugs. $\propto_2$ – adrenoceptors are found in many sites throughout the CNS, but the highest densities are found in the locus ceruleus, the predominant noradrenergic nuclei of the brainstem which is an important modulator of vigilance. Presynaptic activation of $\propto_2$ Aadrenoceptor in the locus ceruleus inhibits nor epinephrine (NE) release and results in sedative and hypnotic effects. Locus ceruleus is the site of origin for descending medullospinal noradrenergic pathway which is an important modulator of nociceptive neuro transmission. Stimulation of the $\propto_2$ –adrenoceptors in this area terminates mainly the propagation of pain signals leading to analgesia. Post synaptic activation of $\propto_2$ – adrenoceptors in the CNS causes decrease in sympathetic activity which leads to hypotension and bradycardia. Also cardiac vagal activity is augmented and all the effects together produce analgesia, sedation and anxiolysis.

At the spinal cord, stimulation of $\propto_2$ –receptors at the substantia gelatinosa causes inhibition of the firing of nociceptive neurons and inhibition of release of substance P.$\propto_2$adrenoceptors also have analgesic mechanisms by preventing NE release at the nerve endings whereas the

spinal mechanism is the principal mechanism for the analgesic action, but clear evidence exists for both supraspinal and peripheral sites of action[16]. $\propto_2$ - receptors located on blood vessels mediates vasoconstriction whereas those located on sympathetic terminals inhibit NE release. In other areas these $\propto_2$ adrenoceptors cause contraction of vascular and other smooth muscles, decreased salivation, decreased secretion and decreased bowel motility in the gastrointestinal tract, and also it causes inhibition of renin release, increased glomerularfiltration, increased sodium and water in the kidney, decreased insulin release from pancreas, decreased intraocular pressure, decreased platelet aggregation and decreased shivering threshold by 2^oC[17].

Pharmacokinetics: Absorption and distribution:

Dexmedetomidine in the dose range of 0.2 to 0.7 μg/kg /hr exhibits linear pharmacokinetics and it is administered as intravenous infusion upto 24 hours. Also the distribution phase is rapid, its half life of distribution is approximately 6 minutes, and eliminationhalf life is 2 hours.

The steady-state volume of distribution is 118L Average protein binding is 94%. Context- sensitive half life ranges from 4 minutes after a 10-minute infusion to 250 minutes after an 8-hour infusion. Its oral bioavailability is poor, which is because of extensive first-pass

metabolism. The bioavailability of sublingual route is high (84%) and it offers a potential role in pediatric sedation and premedication[18].

Metabolism and excretion

Dexmedetomidine undergoes biotransformation through direct N-glucuronidation and cytochrome P-450 (CYP 2A6) mediated aliphatic hydroxylation to its inactive metabolites. Metabolites are excreted in the urine(95%) and in the feces (4%). Dose has to be reduced in patients with hepatic failure.

Pharmacodynamics of Dexmedetomidine

∝- adrenoceptor agonists have different $\propto_2$ / $\propto_1$ selectivity.$\propto_2$ / $\propto_1$ selectivity of dexmedetomidine is 1620:1 whereas it is low for clonidine and hence dexmedetomidine is 8 times more powerful $\propto_2$ – adrenoceptor than clonidine.

CVS:

Dexmedetomidine does not have any direct effects on the heart. It causes a dose dependent increase in coronary vascular resistance and oxygen extraction and the supply / demand ratio is unaltered. It evokes a biphasic blood pressureresponse. A short hypertensive phase and subsequent hypotension and the 2 phases are mediated by 2 different $\propto_2$ – AR Subtypes: the ∝-2B AR is responsible for the initial hypertensive phase, hypotension is mediated by the ∝2A –AR[19]. Younger patients

with high level of vagal tone develop bradycardia and sinus arrest which were effectively treated with anticholinergic agent.

RS:

Dexmedetomidine does not produce respiratory depression even at high doses. It can be safety used in spontaneously breathing ICU patients after surgery. It maintains sedation without cardiovascular instability or respiratory drive depression. Hence it is used during weaning and extubation in trauma / surgical ICU Patients in whom previous attempts at weaning have failed because of agitation associated with hyperdynamic cardio pulmonary response[20].

CNS:

Dexmedetomidine reduces cerebral blood flow and cerebral metabolic requirement of oxygen. Dexmedetomidine enhances cumulative performance and also possess sedative, analgesic and anxiolytic action through $\propto_2$ –AR[21] .It reduces levels of circulating and brain cetecholamines, thus balancing the ratio between cerebral oxygen supplies and reduces excitotoxicity, improves the perfusion in the ischemic penumbra, hence it possess excellent neuroprotective action. In subarachnoid haemorrhage it reduces the levels of glutamate which in responsible for cellular brain injury.

Endocrine and renal effects

Dexmedetomidine activates peripheral presynaptic $\propto_2$–AR, thus catecholamine release is reduced and hence sympathetic response to surgery is also reduced. It is an imidazole agent but doesnot inhibit steroidogenesiswhen used as an infusion for shortterm sedation[22].

Adverse Effects:

Side effects reported are hypotension, hypertension, nausea, vomiting, dry mouth, bradycardia, atrial fibrillation, pyrexia, chills, pleural effusion, atelectasis, pulmonary edema, hyperglycemia, hypocalcaemia, acidosis, etc., Transient hypertension is produced when dexmedetomidine infusion is rapidly administered (Loading dose of 1μg/Kg / hr if given less than 10 minutes) and this is mediated by peripheral ∝2B –AR vasoconstriction.

The occurrence of Hypotension and bradycardia is mediated by central ∝2A-AR, causing decrease of noradrenaline release from the sympathetic nervous system. Supersensitization and up regulation of receptors occur during long term use, hence abrupt discontinuation not advised. Withdrawal syndrome of nervousness, agitation, headache and hypertensive crisis occur during abrupt discontinuation.

Clinical applications of dexmedetomidine premedication

Dexmedetomidine is used as an adjuvant for premedication since this drug possess sedative, anxiolytic, analgesic, sympatholytic, and stable humodynamic profile. Premedication dose is 0.33 to 0.67 mg /kg IV given 15 minutes before surgery. Oxygen consumption is decreased in intraoperative period and in post operative period[23].

Intra operative use:

Dexmedetomidine attenuates the homodynamic stress response which occurs during intubations and extubation by sympatholysis[24]. Dexmedetomidine potentiates anaesthetic effect of all the anaesthetic agents, thus reducing their requirement.

Loco regional analgesia

Highly lipophilic nature of dexmedetomidine facilitates rapid absorption into the cerebrospinal fluid. It binds to $\propto_2$ – AR of spinal cord for its analgesic action. Sensory and motor block produced by local anesthetics is prolonged. It is also used in intravenous regional anesthesia (IVRA), brachial plexus block and intraarticularly. It is also given through intraarticular route in arthroscopic knee surgeries to improve the duration of postoperative analgesia.

Sedation in ICU

Dexmedetomidine produce cooperative sedation. It does not interfere with the respiratory drive hence it facilitates early weaning from

ventilator, thus reducing ICU stay costs. Many studies have recommended their use for longer than 24 hrs[25]. Their other beneficial effects are analgesic sparing effects, reduced delirium and agitation, minimal respiratory depression and desirable cardio vascular effects.

Procedural sedation

Dexmedetomidine is used for short term procedural sedation like transesophageal echocardiography, colonoscopy, awake carotid endarterectomy, shockwave lithotripsy, elective awake fiberoptic intubation[26],pediatric MRI. The dose is 1 μg/kg which is followed by an infusion of 0.2μg/kg/h.

Controlled hypotension

Spinal fusion surgery for idiopathic scoliosis, septoplasty and tympanoplastyoperations and maxillofacial surgery have been done with dexmedetomidine induced hypotension.

Analgesia

Dexmedetomidine activates ∝2 –AR in the spinal cord, thus the transmission of nociceptive signals is reduced. It possesses significant opioid sparing effect.

Cardiac surgery

Dexmedetomidine reduces the extent of myocardial ischemia during cardiac surgery. Its other uses are in the management of

pulmonary hypertension in patients undergoing mitral valve replacement[27].

Neurosurgery

Dexmedetomidine possess neuro protective effect. It also attenuates delirium and agitation, so that postoperative neurological evaluation will be easier. It has a role in functional neurosurgery like awake craniotomy surgeries and implantation of deep brain stimulators for Parkinson's disease[28].

Obesity:

In morbidly obese patients this drug does not cause respiratory depression in the dose of 0.7µg /kg intra operatively.

Obstetrics

Dexmedetomidine is also used in obstetrics due to its maternal hemodynamic stabilizing property. It also produces anxiolysis and stimulation of uterine contractions. Since it is highly lipophilic it does not cross placenta and hence it cause less chance of fetal bradycardia.

Pediatrics

Recently it is used in pediatric patients for sedation during non-invasive procedures in radiology like CT scan and MRI [29].

Other uses

Used as an anti-shivering agent Used in the treatment of withdrawal from benzodiazepines, opioids and alcohol.

REVIEW OF LITERATURE

1. SubhiM Al-Ghanam et al(2009)[30] studied the effect of adding Dexmedetomidine versus fentanyl to intrathecal 0.5% isobaric bupivacaine on spinal characteristics in gynecological procedures. This double blind prospective study was conducted in 78 patients and half of them received Dexmedetomidine 5µg and the remaining half received 25µg fentanyl with 10mg isobaric Bupivacaine.It was found that the mean time of sensory regression to S1 and also the regression of motor block was significantly longer in Dexmedetomidine group.Hence it was concluded that when comparing to 25µg of fentanyl,5µg of Dexmedetomidine seems to be an attractive alternative as an adjuvant to spinal bupivacaine with only a very minimal sideeffects and excellent quality of spinal analgesia in gynecological procedures.
2. Rajni Gupta et al(2011) [31]studied the effect of adding Dexmedetomidine with isobaric Ropivacaine for post operative analgesia.This randomized double blind study was conducted in sixty patients divided into two groups and one group received 3 ml of 0.75% isobaric Ropivacaine with 0.5 ml normal saline,other group received 3 ml of 0.75% isobaric Ropivacaine with 0.5 ml of Dexmedetomidine5µg. Their study showed that mean time of regression to S1 and the duration of analgesia was significantly

prolonged in Dexmedetomidine group. It was concluded that the addition of Dexmedetomidine to Ropivacaine intrathecally produces a prolongation in the duration of motor as well as sensory block.

3. Rajni Gupta et al (2011)[32] studied the comparative effect of intrathecal Dexmedetomidine and fentanyl as adjuvants to bupivacaine.They have found that intrathecal Dexmedetomidine is associated with prolonged motor and sensory block, hemodynamic stability and reduced need for rescue analgesia in 24 hrs as compared to fentanyl.
4. Mahmoud M Al-Mustafa et al(2009)[33]studied the effect of different doses of Dexmedetomidine when added to spinal isobaric bupivacaine for urological procedures.This study was conducted in 66 patients who were randomly assigned into three groups.The first group received Bupivacaine 12.5 mg with saline, the second group received 12.5 mg Bupivacaine with 5µg of Dexmedetomidine, and the third group received 12.5mg Bupivacaine with 10µg of Dexmedetomidine.Itwas observed that the onset of sensory and motor block was significantly faster and duration of sensory and motor block was significantly prolonged in Dexmedetomidine group in a dose dependent manner.Hence it was concluded that Dexmedetomidine has a dose dependent effect on the onset and regression of sensory and motor block when it is added as adjuvant to Bupivacaine in spinal anesthesia.

5. Hala E A Eid et al(2011)[34]studied the dose related effect of intrathecal Dexmedetomidine when added to hyperbaric Bupivacaine .This double blind prospective randomized study was conducted in forty eight patients who were scheduled for anterior cruciate ligament reconstruction.First group received 10µg of Dexmedetomidine, the second group received 15µg ofdexmedetomidine and third group received normal saline with 3ml of 0.5% bupivacaine.It wasfound that the two segment regression, sensory regression to S1,regression of motor block to modified bromage 0,time to first rescue analgesia was prolonged significantly with Dexmedetomidine.Also it was associated with decreased post-operative pain score.Hence it was concluded that intrathecal Dexmedetomidine in doses of 10µg and 15µg causes prolongation of anaesthetic and analgesic effects of spinal hyperbaric bupivacaine in a dose dependent manner.
6. Ashraf Amin Mohamed et al[35] studied the comparison of analgesic efficacy of intrathecally administered Dexmedetomidine or Dexmedetomidine combined with fentanyl in patients undergoing major abdominal cancer surgery. This double blind randomized study was conducted in ninety patients who received intrathecally 10 mg bupivacaine 0.5%(control group)or 10mg bupivacaine0.5% and 5µg Dexmedetomidine (Dexmedetomidine group)or 10mg bupivacaine 0.5%and 5µg Dexmedetomidine and 25µg fentanyl

(Dexmedetomidine plus group).It was concluded that Dexmedetomidine 5µg when given intrathecally improves the quality and the duration of postoperative analgesia and also it provides an analgesic sparing effect in patients undergoing major abdominal cancer surgery and it was proved that fentanyl has no valuable clinical effect.

7. KANAZI et al studied[36], the effect of low dose Dexmedetomidine or clonidine on the characteristics of bupivacaine spinal block.This randomized prospective double blind study was conducted in sixty patients undergoing transurethral resection of prostate or bladder tumour under spinal anesthesia. The patients were randomly allocated into one of three groups. Group B received 12mg of hyperbaric bupivacaine,group D received 12mg of bupivacaine supplemented with 3µg of Dexmedetomidine and group C received 12mg of bupivacaine supplemented with 30µg of clonidine. It was concluded that when Dexmedetomidine 3µg or clonidine 30µg when added to intrathecal bupivacaine causes a similar prolongation in duration of the sensory and motor blockade with preserved hemodynamic stability and lack of sedation.
8. DeepikaShuklaet al[37] conducted a comparative study of intrathecal dexmedetomidine with intrathecal magnesium sulphate when used as adjuvants to bupivacaine. This prospective randomized double-blind

study was conducted in 90 patients to evaluate the onset and duration of sensory and motor block and also the peri operative analgesia and adverse effects of Dexmedetomidine and magnesium sulphate when given intrathecally with 0.5% hyperbaric bupivacaine for spinal anesthesia. It was concluded that onset of anesthesia was rapid and of prolonged duration in the Dexmedetomidine group when compared to magnesium sulphate group and the groups were similar with respect to hemodynamic variables and there were no side effects in either of them.

9. Anand et al [38]studied the effects of caudal Dexmedetomidine combined with ropivacaine to provide post-operative analgesia in children and also established its safety in pediatric population. This double blind, randomized, prospective, parallel group study was conducted in 60 children who were allocated into two groups. Group RD received 0.25% ropivacaine 1ml/kg with Dexmedetomidine 2µg/kg making the volume to 0.5 mg and Group R received 0.25% ropivacaine 1ml/kg + 0.5 ml normal saline.Induction done with 50% N_2O and 8% sevoflurane in oxygen with spontaneous ventilation. It was concluded that caudal Dexmedetomidine (2µg/kg) with 0.25% ropivacaine (1 ml/kg) for pediatric lower abdominal surgeries result in significant post-operative pain relief and better quality of sleep and a

prolonged duration of arousable sedation with less incidence of emergence agitation.

10. Ibrahim F A Khalifa[39] conducted a study in fifty ASA grade I&II patients, who were scheduled for elective inguinal hernia repair. 25 patients in Group D received Dexmedetomidine 0.5 ml and group S patients received sufentanil 0.1 ml + normal saline 0.4 ml added to 2 ml heavy bupivacaine. They concluded that the addition of Dexmedetomidine 5µg and sufentanil 5µg intrathecally provide improved post operative analgesia and better hemodynamic stability. Also it was found that 5µg Dexmedetomidine seems to be an attractive alternative as adjuvant to spinal bupivacaine in prolonged surgical procedures with minimal side effects and excellent quality of spinal analgesia.
11. A E Kyles et al[40] studied that intrathecal administration of the α2-adrenoceptor agonists, clonidine, xylazine, guanfacine and Dexmedetomidine produced dose dependant antinociception in the rat. These studies also demonstrate that a significant proportion of the antinociceptive effect of systemically administered xylazine is mediated by spinal α2-adrenoceptors.
12. AnjuGrewal et al[41] reported the experimental animal and human studies of intrathecal Dexmedetomidine added as an additive to local anesthetics, and found that there is a dose dependent prolongation of

sensory block, increase in motor block, along with prolongation of the post operative analgesia, thus reducing the dose requirement in high risk group of patients.

13. Lawhead R G et al[42] suggested that the predominant α2-adrenergic sub type present in human spinal cord is the α-2A sub type and the α2 –adrenergic receptor density was found to be significantly greater in the sacral region of the cord as compared to lumbar or thoracic regions.
14. L Hennawyet al[43] studied that the addition of clonidine or Dexmedetomidine to bupivacaine prolongs caudal analgesia in children.
15. Tatsushiet al[44] have concluded that all α2- adrenoceptor agonists enhance the degree of local anesthesia of lidocaine in a dose-dependent manner and suggested that Dexmedetomidine acts through α-2A adrenoceptors.
16. Eisenach,Dekock et al[45] have studied that when clonidine is administered intrathecally with bupivacaine it cause prolongation of sensory and motor block.
17. KritonS.Filos, et al[46] evaluated the dose-response hemodynamic and analgesic profiles of intrathecal clonidine after a surgical intervention,without perioperative administration of other analgesics.This study was done in 3 groups of patients who received

150,300,450µg clonidine and postoperative analgesia was assessed.It was found intrathecal clonidine decreases pain in all 3 groups in a dose dependent manner with hemodynamic stability.

MATERIALS AND METHODS

Study design:

This was a randomised, prospective, parallel group, double-blinded study.

Randomisation:

Simple randomised sampling was done by computer generated random numbers.

Sample size:

Sixty patients were studied.

INCLUSION CRITERIA:

- Age between 18-60 years of both sexes
- ASA I and II patients
- Elective surgeries (Inguinal herniorrhaphy and Vaginal hysterectomies)

EXCLUSION CRITERIA:

- Known hypersensitivity to any of study drugs
- Known contra indication to Regional Anesthesia
- Known or suspected coagulopathy
- Renal disorders
- Hypertension , IHD , Heart blocks ,Arrhythmias, Cardiac valvular abnormalities

- Patients on β blockers
- Patient on any long term analgesic therapy
- Patient on medications known to interact with study drugs

Allocation:

After obtaining Institutional Research and Ethical Committee (TIREC) approval and written informedconsent, the patients were randomlyallocated into three groups.

1. Group A (n=20)
2. Group B (n=20)
3. Group C (n=20)

Intervention:

Spinal administration of the drug mixture

1. Group A (n=20) – 0.5% hyperbaric bupivacaine 2.4ml (12mg) + dexmedetomidine 5 μg in 0.6 ml normal saline.
2. Group B (n=20) – 0.5% hyperbaric bupivacaine 2.4ml (12mg) + dexmedetomidine 10 μg in 0.6 ml normal saline.
3. Group C (n=20) – 0.5% hyperbaric bupivacaine 2.4ml (12mg) + dexmedetomidine 15 μg in 0.6 ml normal saline.

Masking:

The anesthesiologist who administered the drug and theobserver were blinded to the study. Sterile syringescontaining 3.0 ml of the total volume of the drug wereloaded by another anesthesiologist not

participating in the study. The intraoperativemonitoring and postoperative observation was doneby the same anesthesiologist who administered thedrug, but was unaware of the content of thesyringes.

Pre-anesthetic evaluation:

Patients included in the study underwent thorough pre-operative evaluation which included the following.

HISTORY

History of co-morbid medical illness, any previous history of surgery under anesthesia evaluated.

PHYSICAL EXAMINATION

- General condition of the patient
- Vital signs
- Examination of CVS,RS,CNS and spinal columns
- Airway assessment
- Investigations like Hb%, BT, CT, Random blood sugar, BloodUrea, SerumCreatinine, Chest X ray, ECG, Bloodgrouping&typing done

Emergency drugs and equipmentswere kept ready. Pre-loading done with 20 ml/kg of intravenous infusion of Ringer lactate. Monitors were connected to the patients and baseline values of heart rate,systolic,diastolic,mean arterial pressure,oxygen saturation were noted.

Under strict aseptic precaution the sub-arachnoid block was done approaching through L3-L4 interspacewith a 26 gauge quincke's needle. After confirming free flow of CSF, the drug was injected according to the group assigned. After injecting the drug, the patients were turned to supine position. When the peak level of sensory block is reached, the surgeon was instructed to proceed.

The following parameters were recorded:

Pulse rate, systolic blood pressure, mean blood pressure, diastolic blood pressure, respiratory rate, SPO2 were recorded before starting procedure and thereafter 5, 10, 15, 20, mins interval till the end of the surgery and thereafter at hourly second and fourth hourly interval till 24 hours. Hypotension was defined as systolic blood pressure less than 90mm Hg or decrease in MAP below 20%of the baseline value. Hypotension, if any occurred was treated with Inj.Ephedrine(6mg) incremental boluses.

SENSORY BLOCKADE:

Sensory blockade was assessed by pin prick with a short hypodermic needle at 1 minute interval until the block reached T10 level and the maximum height of the sensory block was noted at 20 minutes.Onset of sensory blockade was defined as the time taken from the drug injection to the time to reach T10 level and the offset of sensory block was presumed when pin prick sensation at S1 dermatome has

returned.Duration of sensory block was defined as the time interval elapsed between onset of sensory block at T10 to regression of sensory block to S1.

MOTOR BLOCKADE:

Assessed using Modified Bromage score.

GRADE

0 - No motor block

1 - Inability to raise extended legs

2 - Inability to flex knee joints

3 - Inability to flex ankle joints

This was assessed at 1 minute interval until complete motor block occurred. Onset of motor block was defined as the time taken from the injection of drug to the development of complete motor blockade, i.e., Bromage score-3. Complete recovery from motor block was defined as attaining Bromage score-0 and the duration of motor block means that the time taken from the onset of complete motor blockade to complete recovery of motor block.

ASSESSMENT OF PAIN:

Pain was evaluated using Visual Analogue Scale.

0-1 Excellent

2-4 Good

5-6 Fair

7-8	Poor
9-10	No relief

Inj. Diclofenac 75mg was administered intra-muscularly as a rescue analgesic when the pain score crossed a score of 4.

Duration of Analgesia:

It is the period from the time of subarachnoid block to the time when the patient needs the first dose of rescue analgesic drug.

SEDATION:

Assessed using Ramsay Sedation Score.

Grade	Description
1	Anxious and agitated
2	Cooperative and tranquil
3	Drowsy but responsive to command
4	Asleep but responsive to a glabellar tap
5	Asleep with a sluggish response to tactile stimulation
6	Asleep and no response

Post operatively the patients were followed for upto 24 hrs for any adverse effectslike nausea, vomiting, pruritus,respiratory depression,any neurological complications and urinary retention.

OBSERVATION AND RESULTS

Statistical Analysis:

The statistical procedures were performed by the statistical package IBM SPSS statistics - 20. The P - values less than 0.05 ($P<0.05$) were treated as significant in two tail condition. The Randomization of three groups was done by matching their ages, demographic factors and hemodynamic factors such as pulse rate, SBP, MAP SPO2, and duration of surgeryby ANOVA (Analysis of Variance). The differences between them were interpreted by the Post hoc test of Bonferroni. Similarly, the onset time for sensory block, and motor blocks were compared between groups by ANOVA. The intra and post-operative pulse rates, SBP, MAP and SPO_2 at different intervals were compared between groups by ANOVA and interpreted the difference by Post hoc test of Bonferroni. The sensory level and sedation score between three groups were analyzed and interpreted by χ^2 test (Chi- square). The duration of analgesia between the groups were analyzed and interpreted by Kaplan- Mayer Survival Function.

RESULTS:

Randomization by group matching:

Table-1.Matching of three groups according to their age.

Variables	Group	n	Mean	S D	ANOVA 'F'	df	Significance
Age	A	20	44.4	10.7	0.249	2, 57	P>0.05
	B	20	46.0	5.6			
	C	20	44.4	8.1			

The three groups were matched according to their age for randomization and found that there was no difference between the mean ages between them (44.4 ± 10.7 ≈46.0±5.6 ≈ 44.4±8.1 and P>0.05).

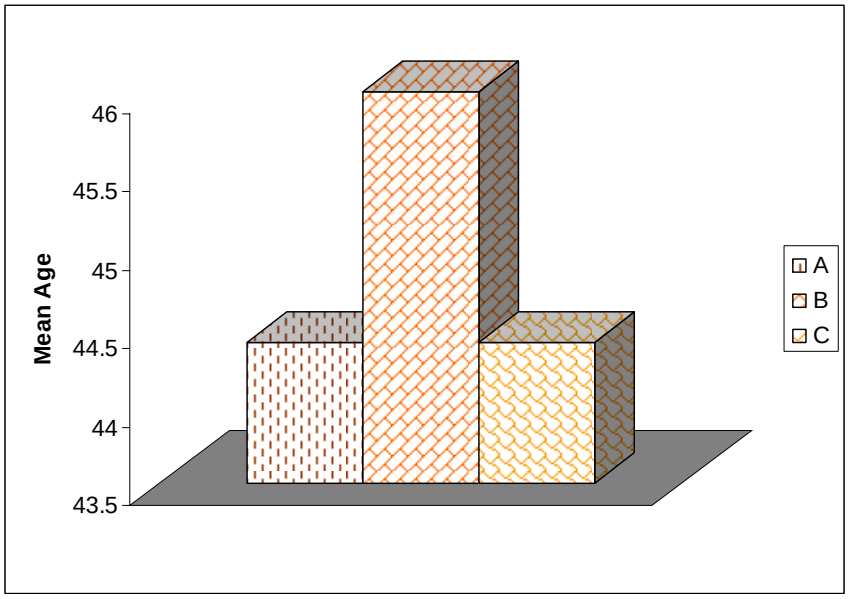


Table-2.

Matching of three groups according to their preoperative hemodynamic Characteristics and duration of surgery.

Variables	Group	n	Mean	S D	ANOVA 'F'	df	Significance
Pre Pulse rate	A	20	82.2	4.4	2.015	2, 57	.143
	B	20	85.7	7.3			
	C	20	82.4	6.6			
Pre SBP	A	20	120.5	10.9	1.610	2, 57	.209
	B	20	119.9	10.1			
	C	20	125.4	10.7			
Pre MAP	A	20	91.1	6.9	0.620	2, 57	.541
	B	20	89.8	8.1			
	C	20	92.6	9.1			
Pre SPO2	A	20	99.8	0.4	2.375	2, 57	.102
	B	20	99.8	0.4			
	C	20	100.0	0.0			
DOS	A	20	97.6	34.1	0.620	2, 57	.542
	B	20	100.9	30.3			
	C	20	108.1	26.1			

Table – 2 shows the haemodynamic variables and the duration of surgery of three groups. The mean pre-op pulse rate, SBP, MAP, SPO2 and duration of surgery were matched and found that no significant differences were observed between the three groups (P>0.05).

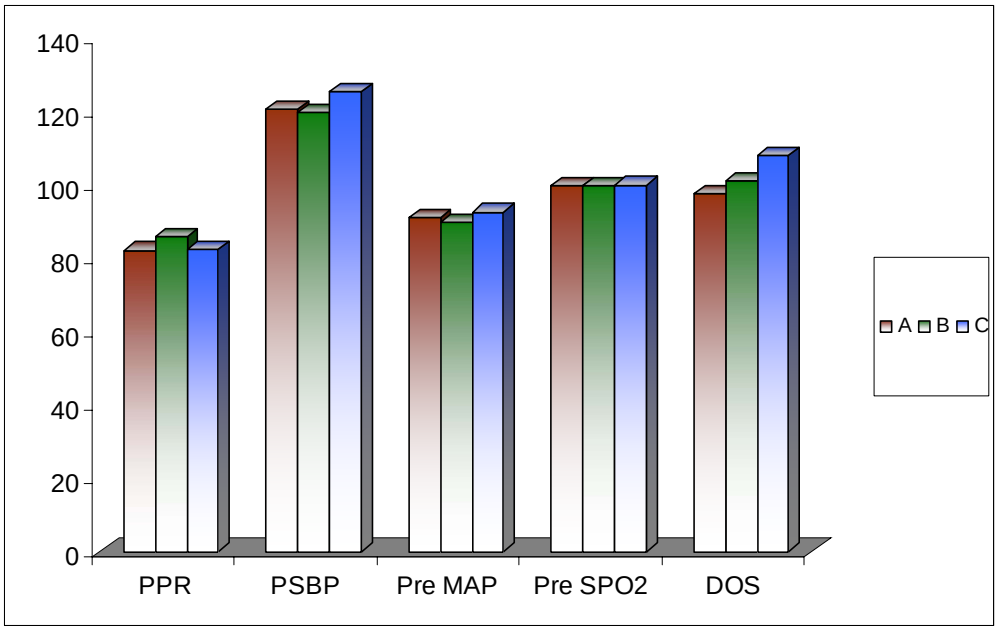


TABLE - 3

Comparison of onset of blockade between groups.

Block	Groups	N	Mean (Sec.)	SD	ANOVA 'F'	df	Sig (P)
OTSB	A	20	226.1	28.7	8.903	2, 57	.000
	B	20	206.8	14.9			
	C	20	197.2	20.2			
OTMB	A	20	233.0	23.3	31.733	2, 57	.000
	B	20	228.2	16.8			
	C	20	190.4	14.2			

Table – 3 shows the comparison of onset of sensory and motor blockade between the three groups. The mean on set time of A group was significantly higher than the other two groups B&C (A>B&C; 226.1±28.7>206.8±20.2&197.2±14.9 and P<0.05). The mean values of B& C groups were approximately equal (206.8±20.2≈197.2±14.9 and P>0.05).

The mean onset time of motor blockade of C group was significantly lower than the other two groups (190.4±14.2< 233.0±23.3 & 228.2±16.8 and P<.001). The onset time for motor block of the other two groups namely A and B were not significant (233.0±23.3 ≈ 228.2±16.8 and P>0.05).

Comparison of Onset time of Sensory and Motor blocks between groups:

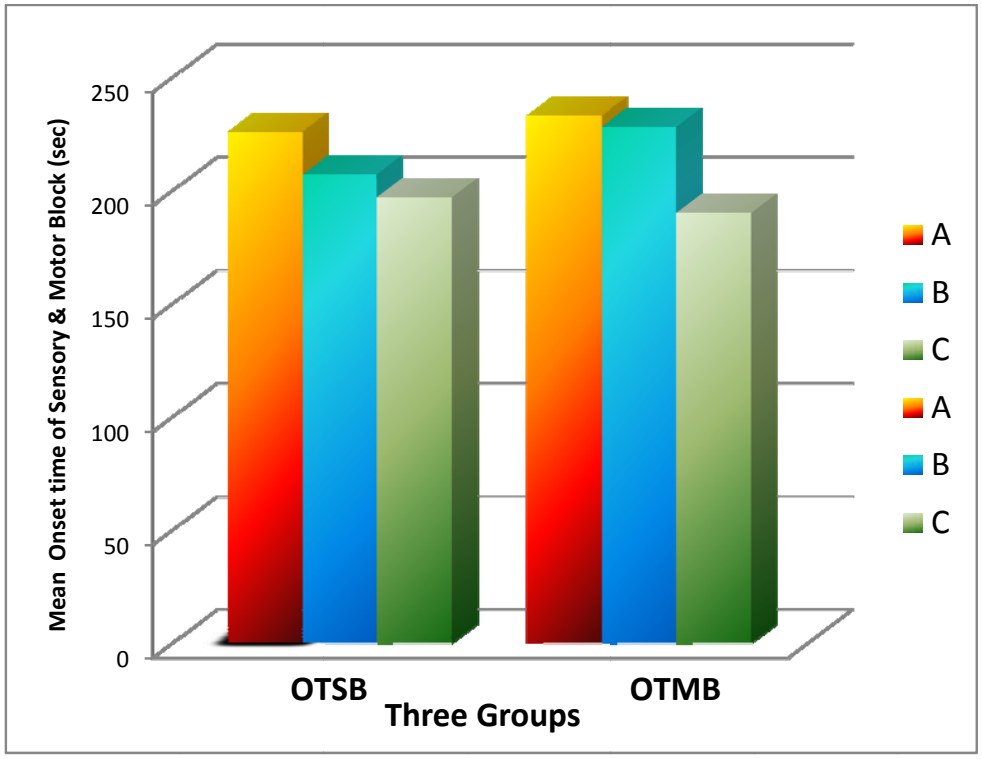
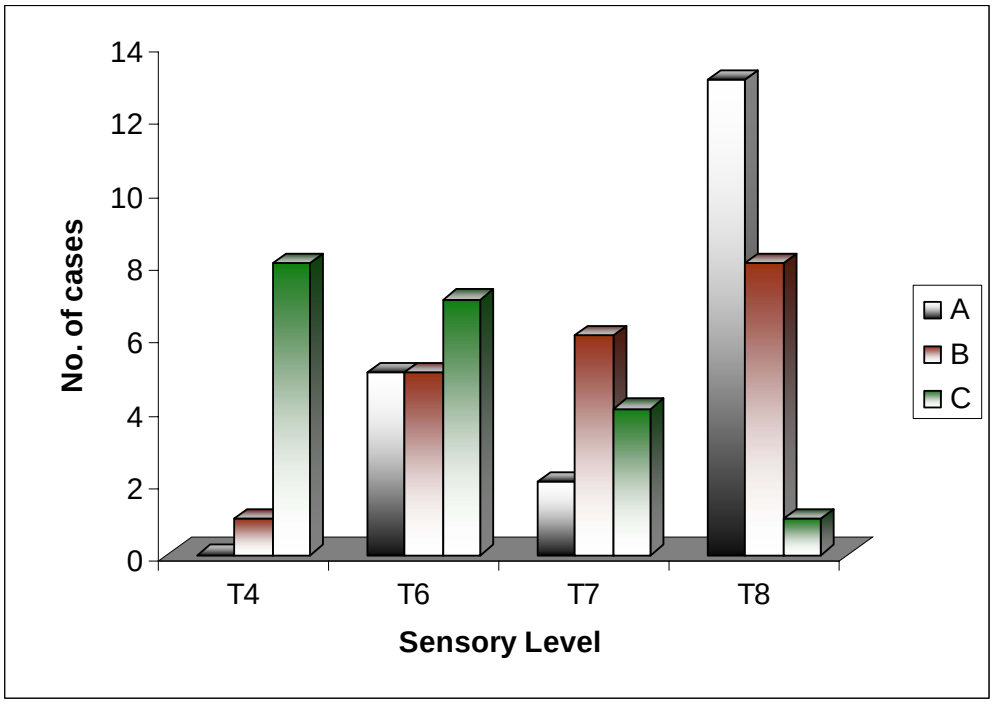


TABLE 4

Level of Blockade: Comparison of Maximum Sensory levels of three groups.

Sensory level	A	B	C	Total	χ^2	df	Sig
T4	0	1	8	9			
T6	5	5	7	17			
T7	2	6	4	12	25.046	6	.000
T8	13	8	1	22			
Total	20	20	20	60			

The level of sensory blockade is compared between the three groups in Table 4.The highest sensory level achieved for A group was T6 and C group was T4.Among the A&B group subjects, 65% and 40% were associated with T8 sensory level and among the C group subjects 40% were associated with T4 sensory level. The above levels were statistically very highly significant (P<0.001).



Comparison of PR, SBP, MAP, & SPO2 at different time between groups:

Table-5.Comparison of Pulse Rate between groups at different time interval.

Pulse rate	Group	n	Mean	S D	F	Df	Sig (P)
5 Minutes	A	20	79.9	3.7	0.481	2, 57	.621
	B	20	81.8	6.4			
	C	20	80.3	8.3			
10 Minutes	A	20	78.3	3.2	1.619	2, 57	.207
	B	20	82.3	7.6			
	C	20	78.5	10.8			
15 Minutes	A	20	76.6	3.4	2.872	2, 57	.065
	B	20	83.9	11.7			
	C	20	78.0	12.7			
30 Minutes	A	20	78.1	4.5	1.345	2, 57	.269
	B	20	81.2	9.8			
	C	20	76.5	11.9			
1 Hour	A	20	75.8	4.2	1.477	2, 57	.237
	B	20	80.7	9.9			
	C	20	77.1	12.0			
2 Hours	A	20	75.6	4.1	2.825	2, 57	.068
	B	20	81.7	8.3			
	C	20	76.9	11.4			

3 Hours	A	20	75.7	4.1	2.428	2, 57	.097
	B	20	81.4	6.8			
	C	20	77.9	11.8			
8 Hours	A	20	76.5	6.1	1.368	2, 57	.263
	B	20	81.2	8.7			
	C	20	78.3	11.4			
12 Hours	A	20	77.3	5.7	1.472	2, 57	.238
	B	20	81.8	8.6			
	C	20	79.2	10.2			
18 Hours	A	20	79.1	5.0	0.640	2, 57	.531
	B	20	82.0	8.6			
	C	20	79.6	11.1			
24 Hours	A	20	79.7	6.2	0.342	2, 57	.712
	B	20	82.0	8.7			
	C	20	80.6	10.9			

The Pulse rates at different intervals such as at 5,10,15,30 minutes, 1, 2, 3,8,12, 18 and 24 hours are shown in the above Table-5. The mean pulse rates at the above different times between the three groups are not significantly different (P>0.05).

Comparison of Pulse Rate between groups at different time interval

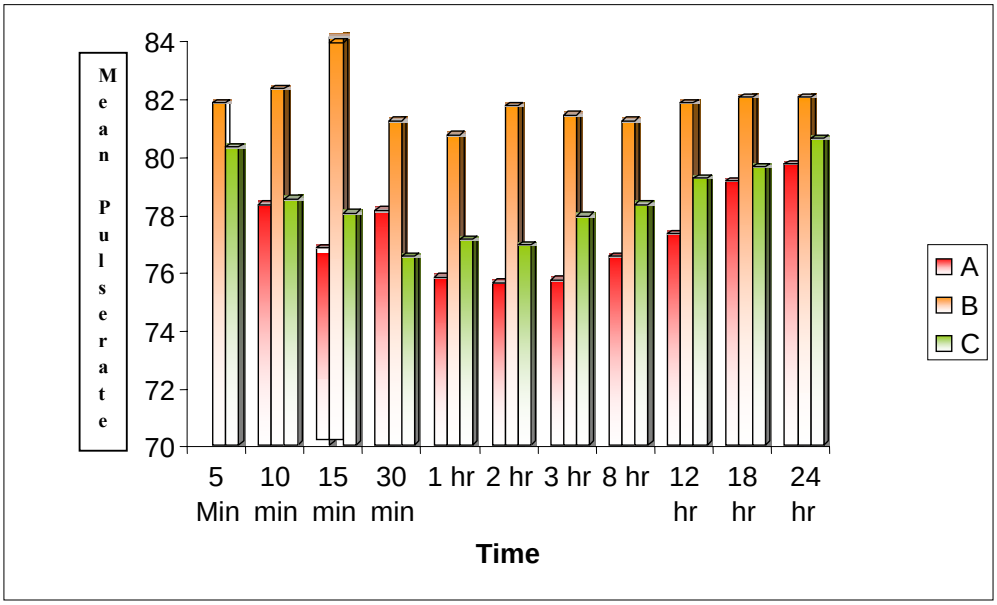


TABLE-6.

Comparison of SBP between groups at different time interval.

SBP	Group	n	Mean	S D	F	df	Sig (P)
5 Minutes	A	20	111.5	10.3	1.723	2, 57	.188
	B	20	110.4	9.4			
	C	20	118.7	22.5			
10 Minutes	A	20	107.0	9.2	2.842	2, 57	.067
	B	20	106.5	7.4			
	C	20	113.8	14.5			
15 Minutes	A	20	102.0	9.5	1.515	2, 57	.228
	B	20	102.5	8.5			
	C	20	107.8	15.6			
30 Minutes	A	20	104.0	9.9	3.044	2, 57	.055
	B	20	104.8	9.3			
	C	20	113.1	17.7			

1 Hour	A	20	107.1	15.4	0.073	2, 57	.930
	B	20	108.3	15.1			
	C	20	108.8	12.7			
2 Hours	A	20	111.3	14.8	0.157	2, 57	.855
	B	20	112.3	14.1			
	C	20	109.9	11.6			
3 Hours	A	20	111.3	14.8	0.046	2, 57	.955
	B	20	112.5	14.4			
	C	20	112.6	16.1			
8 Hours	A	20	107.5	7.8	0.084	2, 57	.919
	B	20	108.5	8.1			
	C	20	107.8	7.7			
12 Hours	A	20	109.1	9.5	0.199	2, 57	.820
	B	20	109.7	9.7			
	C	20	108.0	6.1			
18 Hours	A	20	109.1	9.5	0.233	2, 57	.793
	B	20	109.8	9.7			
	C	20	111.0	7.1			
24 Hours	A	20	108.8	9.6	0.597	2, 57	.554
	B	20	109.5	10.5			
	C	20	112.0	8.9			

The SBP at different intervals such as at 5,10,15,30 minutes, 1, 2, 3,8,12, 18 and 24 hours are shown in the above Table-6. The mean SBP at the above different times between the three groups are not significantly different ($P>0.05$).

Comparison of SBP between groups at different time interval.

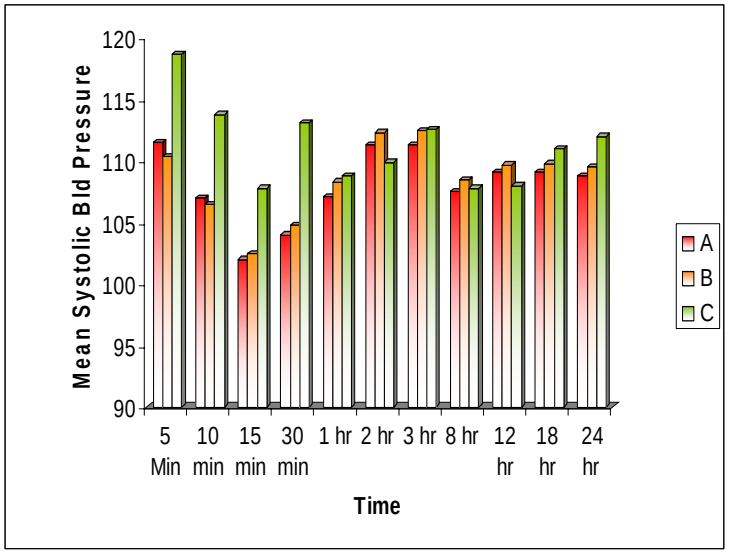


Table-7. Comparison of MAP between groups at different time interval.

MAP	Group	n	Mean	S D	F	df	Sig (P)
5 Minutes	A	20	87.4	6.3	1.001	2, 57	.374
	B	20	86.0	7.3			
	C	20	89.9	11.8			
10 Minutes	A	20	82.6	7.5	0.817	2, 57	.447
	B	20	81.5	7.1			
	C	20	85.4	13.5			
15 Minutes	A	20	80.7	7.8	0.091	2, 57	.913
	B	20	80.0	8.3			
	C	20	81.2	11.3			
30 Minutes	A	20	81.1	7.7	0.388	2, 57	.680
	B	20	81.9	7.7			
	C	20	83.5	10.5			
1 Hour	A	20	81.2	10.3		2, 57	

	B	20	82.5	10.3	0.113		.893
	C	20	81.3	7.3			
2 Hours	A	20	84.3	8.6	0.063	2, 57	.939
	B	20	84.5	8.5			
	C	20	85.3	10.8			
3 Hours	A	20	86.0	8.7	0.054	2, 57	.948
	B	20	87.0	7.9			
	C	20	86.5	10.6			
8 Hours	A	20	84.9	7.8	0.180	2, 57	.836
	B	20	85.9	7.5			
	C	20	84.3	9.3			
12 Hours	A	20	85.5	8.5	0.987	2, 57	.379
	B	20	86.2	8.3			
	C	20	82.9	6.5			
18 Hours	A	20	85.0	8.0	0.549	2, 57	.581
	B	20	85.9	7.7			
	C	20	83.4	7.1			
24 Hours	A	20	85.7	8.0	0.386	2, 57	.681
	B	20	86.7	7.7			
	C	20	84.6	6.7			

The MAP at different intervals such as at 5,10,15,30 minutes, 1, 2, 3,8,12, 18 and 24 hours are shown in the above Table-7. The mean MAP at the above different times between the three groups are not significantly different ($P>0.05$).

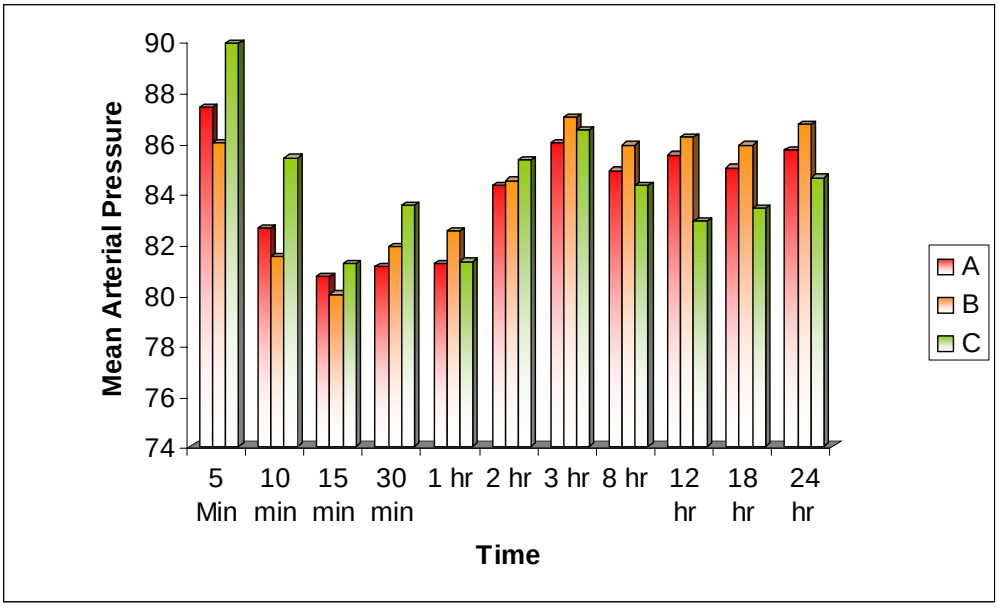


Table-8. Comparison of SPO2 between groups at different time interval.

SPO2	Group	n	Mean	S D	F	df	Sig (P)
5 Minutes	A	20	99.5	0.5	1.354	2, 57	.266
	B	20	99.4	0.6			
	C	20	99.2	0.5			
10 Minutes	A	20	98.8	0.5	2.146	2, 57	.126
	B	20	99.0	0.6			
	C	20	99.2	0.5			
15 Minutes	A	20	98.9	0.6	2.678	2, 57	.077
	B	20	99.0	0.6			
	C	20	99.3	0.4			
30 Minutes	A	20	99.6	0.5	1.004	2, 57	.373
	B	20	99.8	0.3			
	C	20	99.8	0.4			
1 Hour	A	20	99.8	0.4	0.377	2, 57	.687
	B	20	99.9	0.3			

	C	20	99.8	0.3			
2 Hours	A	20	99.8	0.3	0.138	2, 57	.872
	B	20	99.9	0.3			
	C	20	99.8	0.3			
3 Hours	A	20	99.9	0.2	1.000	2, 57	.374
	B	20	100.0	0.0			
	C	20	100.0	0.0			
8 Hours	A	20	100.0	0.0	1.000	2, 57	.374
	B	20	99.9	0.2			
	C	20	100.0	0.0			
12 Hours	A	20	99.9	0.2	1.000	2, 57	.374
	B	20	100.0	0.0			
	C	20	100.0	0.0			
18 Hours	A	20	99.9	0.2	0.500	2, 57	.609
	B	20	99.9	0.2			
	C	20	100.0	0.0			
24 Hours	A	20	99.90	0.2	1.000	2, 57	.374
	B	20	100.0	0.0			
	C	20	100.0	0.0			

The SPO2 at different intervals such as at 5,10,15,30 minutes, 1, 2, 3,8,12, 18 and 24 hours are shown in the above Table-8. The mean SPO2 at the above different times between the three groups are not significantly different (P>0.05).

Comparison of SPO2 between groups at different time interval

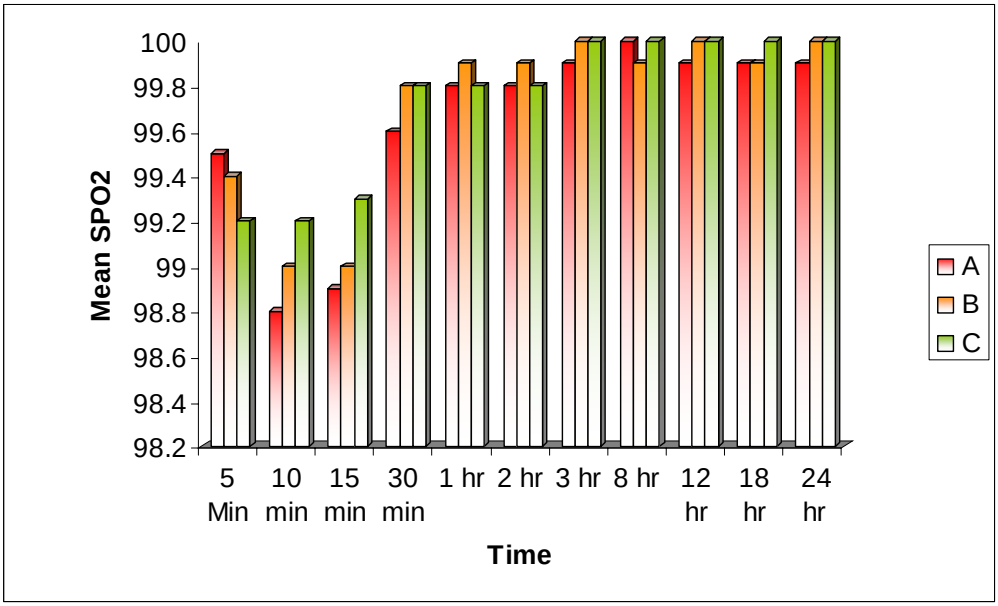


TABLE – 9.Comparison of duration of Sensory and Motor blocks between groups.

Block	Groups	N	Mean (min.)	SD	ANOVA 'F'	df	Sig (P)
DSB	A	20	241.0	48.9	19.371	2, 57	.000
	B	20	290.0	56.2			
	C	20	341.5	47.6			
DMB	A	20	260.6	41.5	52.839	2, 57	.000
	B	20	318.0	31.0			
	C	20	362.5	16.5			

The Duration of Sensory block and Motor blocks are shown in the above Table -9. The duration of sensory block of C group was significantly longer than B and B group was significantly longer than A group (341.5±47.6>290±56.2>241.0 ±48.9 and P<0.001).

Similarly the duration of motor block of C group was significantly longer than B and B group was significantly longer than A group (362.5±16.5>318.0 ±31.0>260.6±41.5 and P<0.001).

Comparison of duration of Sensory and Motor blocks between groups

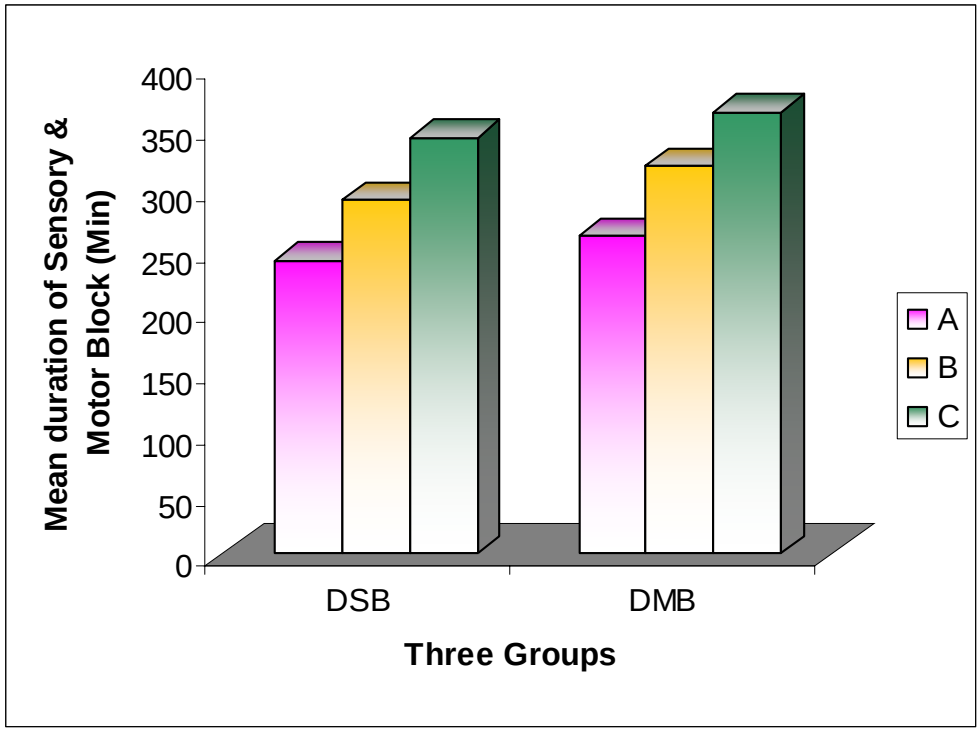


TABLE - 10

Comparison of 2 segment regression time between three groups

Groups	N	Mean	SD	ANOVA 'F'	df	Sig (P)
A	20	139.7	28.2	7.760	2, 57	.001
B	20	143.2	28.8			
C	20	172.7	30.2			

The 2 segment regression time between the three groups is shown in the above table 11. The two segment regression times between three groups were 139.7±28.2, 143.2±28.8 and 172.7±30.2.minutes respectively. The group C regression time was significantly greater than the other two groups (172.7±30.2.> 143.2±28.8 &139.7±28.2, and $P<0.00$). But the regression times between the groups A and B was not significant (143.2±28.8 ≈139.7±28.2 and $P<0.05$).

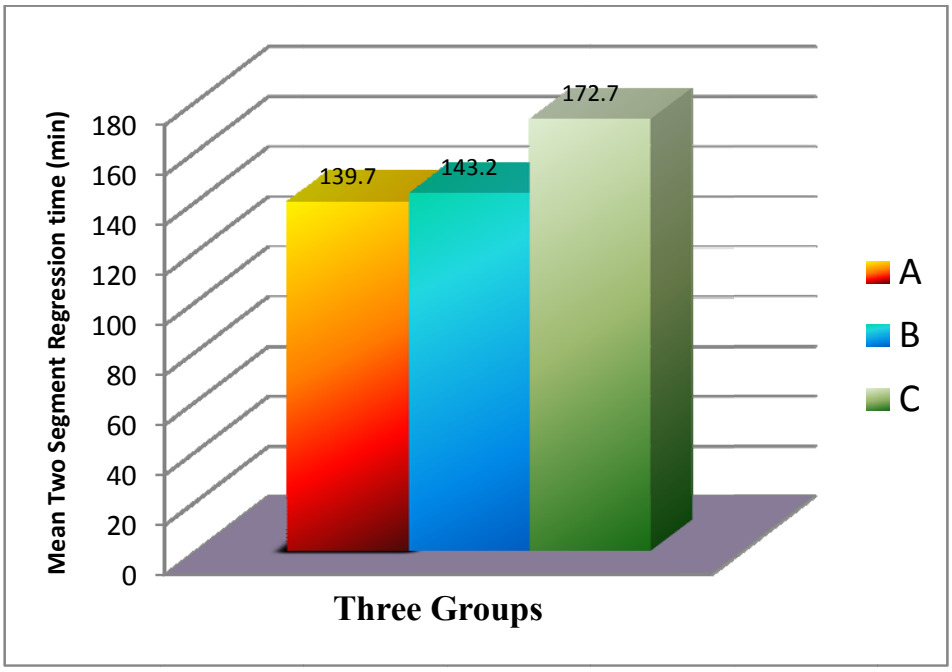


TABLE - 11

Comparison of duration of analgesia between three groups

Groups	N	Mean	SD	ANOVA 'F'	df	Sig (P)
A	20	347.5	101.4	158.851	2, 57	.000
B	20	471.0	24.6			
C	20	740.5	65.9			

The durations of analgesia between the three groups were 347.5± 101.4, 471.0±24.6 and 740.5±65.9 minutes respectively. The mean duration of analgesia of C group was significantly longer than the B group and B group was significantly longer than A group (740.5±65.9>471.0±24.6>347.5± 101.4 and P<0.001).

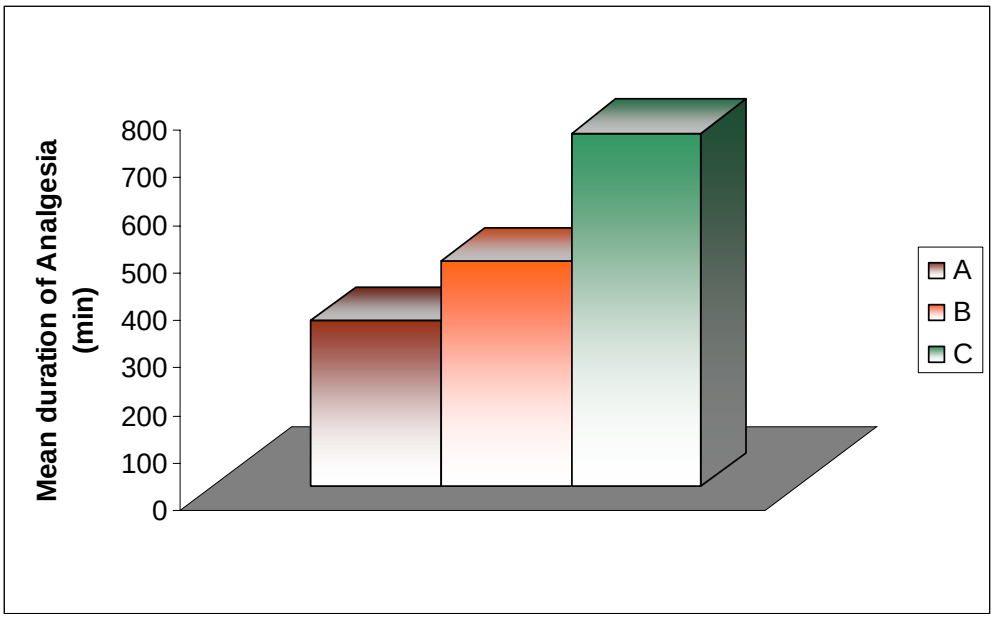
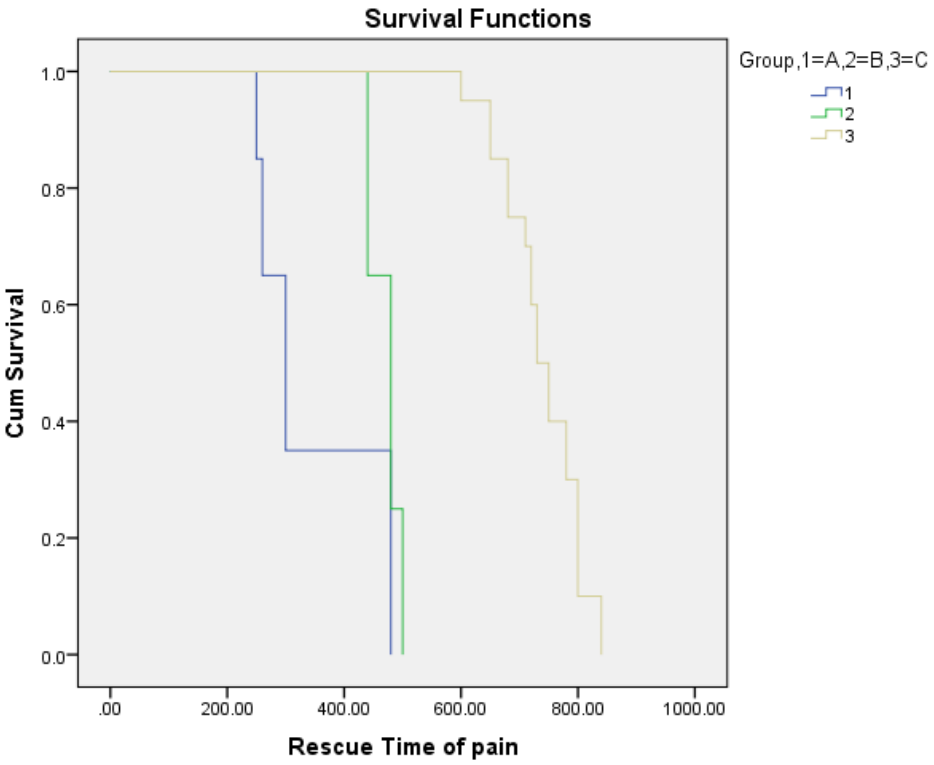


Fig.-1. Comparison of Analgesia survival functions between three groups. (Kaplan Meier Survival function curve)



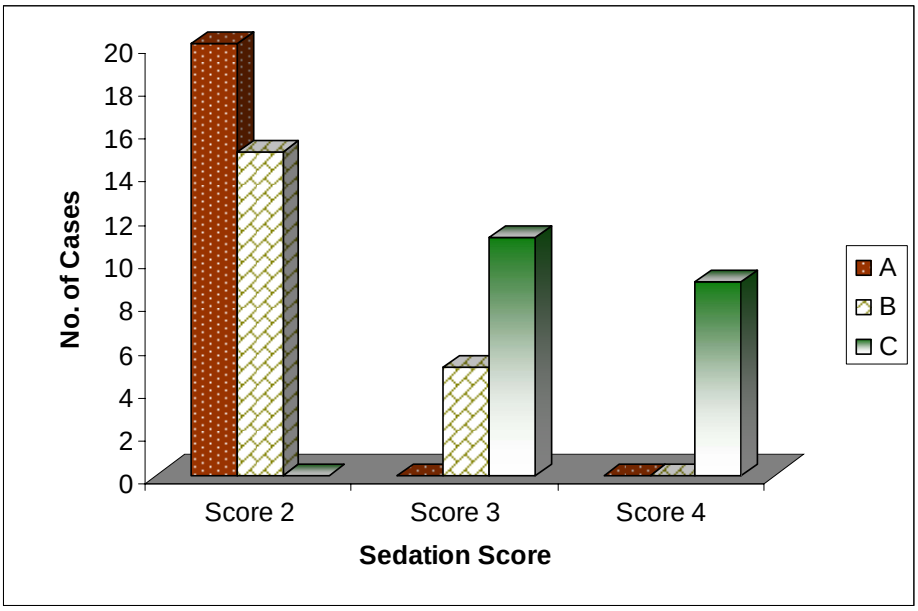
The Fig-1 explains the time of analgesia from the starting of anesthesia to the onset of VAS pain score 4. The range of group A was 250 to 480 minutes, B group was 440 to 500 and C group was 600 to 840 minutes.

TABLE - 12

Comparison of Sedation Score between three groups.

Sedation score	A	B	C	Total	χ^2	Df	Sig
2	20	15	0	35	47.946	4	.000
3	0	5	11	16			
4	0	0	9	9			
Total	20	20	20	60			

The Sedation Score of three groups are compared in Table-12. The highest sedation score achieved for A group was 2 and C group was 4. The C group achieved more sedation levels wherein 55% reached score 3 and 45% reached score 4. The above levels were statistically very highly significant ($P<0.001$).



Adverse effects:

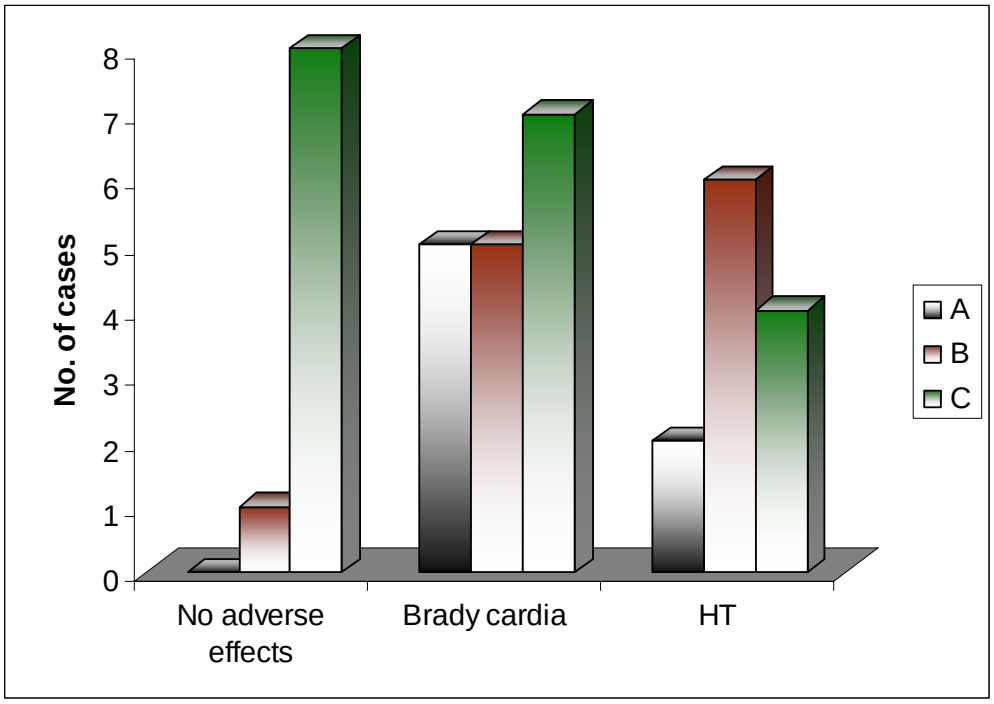
The adverse effects are enumerated between three groups and associated with the respective groups.

Table-13.The adverse effects between the groups.

Adverse effects	**Group,1=A,2=B,3=C**			**Total**	**χ^2**	**Df**	**Sig**
	1	2	3				
Nausea & Vomiting	0	0	0	0	1.780		.776
Pruritus	0	0	0	0			
Respiratory depression	0	0	0	0			
Urinary retention	0	0	0	0			

Brady cardia	1	1	2	4		
Hypotension	1	2	3	6		
Total	2	3	5	10		

The above table-13 associates the adverse effects between the three groups. Bradycardia and Hypotension were the only adverse effects noted, and was much associated with the C group. But the association within the groups did not have any statistical significance (P>0.05).



DISCUSSION

Recent researches have revealed that the administration of an α2-agonist in the centro-neuraxial blockade produces prolonged postoperative pain relief without undue sedation. This effect is due to the sparing of supraspinal CNS sites from excessive drug exposure, resulting in analgesia without heavy sedation. The mechanism by which intrathecal α_2-adrenergic agonists prolong the motor and sensory block of local anesthetics is still not clearly understood. Intrathecal α_2-adrenergic agonists produce analgesia by depressing the release of C-fiber transmitters and by hyperpolarization of post-synaptic dorsal horn neurons. This anti-nociceptive effect may explain the prolongation of the sensory block when added to spinal anaesthetics. The prolongation of the motor block of spinal anaesthetics may result from the binding of α_2-adrenergic agonists to motor neurons in the dorsal horn.

Most of the clinical experience gained in the use of intrathecal α_2-adrenoceptor agonists has been described with clonidine, which has a potent synergistic effect with local anaesthetics. There are only few research available using a combination of intrathecal dexmedetomidine and local anaesthetics. The dose of epidural/ caudal dexmedetomidine reported is in the range of 1.5 - 2 μg/kg.[38] Compared with clonidine, dexmedetomidine has 10 times higher receptor binding affinity. Extrapolations led to the calculation of an equipotent dose of intra-

thecally administered dexmedetomidine. Several clinical studies have established that intrathecal clonidine increases the duration of sensory and motor spinal block when added to spinal local anaesthetics and this effect of clonidine is dose-dependent.[45] Doses of more than 75 μg are accompanied by excessive sedation, hypotension and bradycardia. Intrathecal dexmedetomidine upto 10μg added to local anaesthetics has not produced any major adverse effects during the studies conducted by these authors discussed above. *De kocket al*[45] recommended a dose of 15-45 μg clonidine for supplementation of spinal anesthesia which effectively prolongs the duration of spinal block with minimal sedation and side effects. Higher intrathecal doses of clonidine have been tried. But there is no published literature for intrathecal doses of more than 10μg of dexmedetomidine. The equipotent dose for 5, 10 and 15μg of dexmedetomidine when compared to clonidine would approximate 50, 100 and 150μg respectively.

Kanazi et al,[36] who pioneered using dexmedetomidine in humans for spinal anesthesia, hypothesized that intrathecal dexmedetomidine 3 μg or clonidine 30 μg would be equipotent and would produce a similar effect on the characteristics of bupivacaine spinal anesthesia. These conclusions were arrived, pondering over previous animal studies using intrathecal dexmedetomidine. The authors added a low dose of 3 μg of dexmedetomidine or 30 μg of clonidine to 12 mg of intrathecal

bupivacaine. They found no significant difference between the groups with respect to blockade characteristics, analgesia and sedation. They confirmed their hypothesis that the intrathecal doses of dexmedetomidine and clonidine used in the study are equipotent. *Al-Mustafa et al*,[33] hypothesized that 5 μg and 10 μg of intrathecal dexmedetomidine might be equipotent to 50 μg and 100 μg of intrathecal clonidine respectively. They administered dexmedetomidine intrathecally along with bupivacaine to a maximum dose of 10μg. They observed that dexmedetomidine had a dose dependant effect on the onset and regression of sensory and motor block when used as an adjuvant to bupivacaine in spinal anesthesia. *Ashraf Amin Mohamed et al*,[35] compared 5μg dexmedetomidine with 25μg fentanyl added to bupivacaine for abdominal surgeries. They observed that intrathecal 5 μg dexmedetomidine improved the quality and the duration of postoperative analgesia. In another similar study, *Subhi Al-Ghanemet al*[30]compared the effect of adding 5μg dexmedetomidine versus 25μg fentanyl to intrathecal bupivacaine in vaginal hysterectomies. They concluded that 10 mg plain bupivacaine supplemented with 5 μgdexmetedomidine produces prolonged motor and sensory block compared with 25 μg fentanyl. *Gupta et al*,[31] from their study, concluded that 5μg dexmedetomidine added to ropivacaine intrathecally produces a prolongation in the duration of the motor and sensory block. *Shukla et*

al,[37] compared 10µg of intrathecal dexmedetomidine to magnesium sulphate as adjuvants to bupivacaine, and concluded that dexmedetomidine provided earlier onset and prolonged duration of sensory and motor blockade, without any significant hemodynamic alterations.

In the present study, we observed that the onset time of sensory and motor blockade was dose dependent (*table - 3*). Group A (226.1±28.7 seconds) significantly differed (P<0.001) with group B (197.2±14.9 seconds) & C (206.8±20.2 seconds) in respect of their sensory on set time. This means that the onset of sensory blockade was earlier with higher doses. The onset time of motor block was also earlier with increasing doses. Group C (190.4±14.2 seconds) significantly differed (P<0.001) with group A (233.0±23.3 seconds) & group B (228.2±16.8 seconds). A dose related increase in the level of sensory blockade (C>B>A) was noted (*table - 4*). The duration of sensory and motor blocks (table - 9) between the groups was also dose dependent, and significantly (P<0.001) differed from each other. The duration of both sensory and motor blockade was highest with group C (sensory mean-341.5±47.6 minutes, motor mean-362.5±16.5 minutes). The 2 segment regression time (*table – 10*) was highest with group C (mean-172.7±30.2 mins). The mean duration of analgesia was dose dependent (*table – 11, figure - 1*) with C>B>A (740.5 ±65.9>471.0±24.6> 347.5± 101.4 minutes;

P<0.001). The post-operative sedation (*table – 12*) was also dose dependent with group C exhibiting a minimum score of 3 and maximum of score 4. None of the patient showed signs of respiratory depression.

From the present study, it is clear that intrathecal dexmedetomidine with spinal bupivacaine not only shortens the onset of anesthesia, but also prolongs the duration of blockade and achieves longer duration of analgesia. Adverse effects pertained to opioid administration, such as nausea, vomiting, pruritus and urinary retention were not noted in any of the patient (*table – 13*). Bradycardia and hypotension occurred in 10 out of 60 patients, with group C>B>A (5, 3 and 2 respectively), but were not statistically significant. Bradycardia required no treatment, and correction of hypotension required less than 12-18 mg of ephedrine in incremental boluses. Otherwise the patients remained hemodynamically throughout. The statistical analysis of the pre, intra and post-op hemodynamic variables such as PR, SBP, MAP and SPO_2 between the three groups showed no statistically significant hemodynamic fluctuation. The results of the present study, when compared to the studies of the authors discussed above, have similar outcome with respect to the onset and duration of sensory and motor block, the duration of analgesia and hemodynamic profile.

SUMMARY

To summarize, intrathecal dexmedetomidine added to bupivacaine had a dose dependent effect on the sensory and motor block characteristics showing

1. earlier onset of sensory and motor blockade
2. increased initial segmental level of sensory blockade
3. increased duration of sensory and motor blockade
4. increased duration of post-op. analgesia
5. increased level of sedation

Three different doses (5, 10 and 15μg) did not vary in their effect on the hemodynamic stability or adverse effects.

CONCLUSION

Intrathecal dexmedetomidine added to bupivacaine for lower abdominal surgeries, has a dose dependent effect on the sensory and motor blockade, with earlier onset and increased duration of blockade and prolonged post-operative analgesia, better level of sedation and stable hemodynamics.

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PROFORMA

A COMPARISON BETWEEN THREE DIFFERENT DOSES OF INTRATHECAL DEXMEDETOMIDINE ADDED TO HYPERBARIC BUPIVACAINE FOR INFRA UMBILICAL SURGERIES

Name: Diagnosis:

Age: Procedure:

IP No.: ASA Grade:

Pre-Operative Evaluation:

Pulse: Airway:

BP: CVS:

RR: RS:

Pre-Operative Investigation:

Hb%: Blood Urea:

BT: Sr. Creatinine:

CT: Chest X-ray:

RBS: ECG:

Blood Grouping & Typing:

ANAESTHETIC PLAN

1. Pre-loading

   IV Infusion of Ringer Lactate (20ml/kg)

2. Sub Arachnoid Block

   - Position: Right Lateral - Supine
   - Space: L3-L4
   - Needle size: 26G Quincke's needle
   - No. of attempts & Pass:
   - Paraesthesia: YES / NO
   - Trauma/Bloody tap: YES / NO

3. Drugs

   Group A: 12mg (2.4 ml) of 0.5% hyperbaric bupivacaine + 5 µg of dexmedetomidine in 0.6 ml of NS

   Group B: 12mg (2.4 ml) of 0.5% hyperbaric bupivacaine + 10 µg of dexmedetomidine in 0.6 ml of NS

   Group C: 12mg (2.4 ml) of 0.5% hyperbaric bupivacaine + 15 µg of dexmedetomidine in 0.6 ml of NS

4. Sensory block assessed bilaterally by analgesia to pin-prick with hypodermic needle in mid clavicular line.

Sensory Block:

Onset of sensory block (sec)

Sensory regression time to S1 (min)

Maximum sensory level

Duration of sensory block (min)

Motor Block:

Assessed using modified bromage scale.

Grade

0 -No motor block

1 –Inability to raise extended legs

2 –Inability to flex knee joints

3 –Inability to flex ankle joints

Onset of motor block (sec)

Duration of motor block (min): It is the time from the onset of complete motor blockade to motor regression to bromage scale 0.

5. Duration of Surgery
6. Rescue analgesia – Inj. Diclofenac Sodium – 75 mg IM
7. Duration of analgesia (min)

Assessed using visual analoug scale

0-1 EXCELLENT

2-4 GOOD

5-6 FAIR

7-8 POOR

9-10 NO RELIEF

If pain score is more than 4, supplementary analgesia to be given.

PARAMETERS MONITORED

I. Hemodynamic parameters

Time (min/hr)	PR	SBP	DBP	MAP	SPO_2	RR
0 min						
5 min						
10 min						
15 min 20 min						

30 min						
45 min						
60 min						
90 min						
120 min						
3 hr						
4 hr						
8 hr						
12 hr						
16 hr						
20 hr						
24 hr						

Medication: Inj. Atropine 0.6 mg i.v if PR <60/min.

If systolic blood pressure <90mm Hg or >20% decrease in baseline MAP values; inj. Ephedrine 6mg i.v and fluids given

II. Two Segment Regression Time (min.):

Time to 2 segment regression	

III. Visual Analogue Pain Scale VAS (0-10 point) Scale

Time	VAS Score
2 hr	
3 hr	
4 hr	
8 hr	
12 hr	

IV. Sedation

Assessed using Ramsay Sedation score

Grade	Description
1	Anxious and agitated
2	Cooperative and tranquil
3	Drowsy but responsive to command
4	Asleep but responsive to a glabellar tap
5	Asleep with a sluggish response to tactile stimulation
6	Asleep and no response

Time	Sedation Score
1 hr	
2 hr	
3 hr	
4 hr	
8 hr	
12 hr	

V. ADVERSE EFFECTS

Hypotension	
Bradycardia	
Nausea & Vomiting	
Urinary retention	
Pruritus	
Respiratory depression	

MASTER CHART: GROUP 'A' - BUPIVACAINE WITH 5µg DEXMEDETOMIDINE

							HR																			SPO2																					SBP			
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	ATR OPINE	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN
1	A	ABDUL KHADAR	59	MALE	35988	2	82	80	78	76	80	84	86	80	78	78	76	76	80	80	82	84	88	NIL	100	99	98	100	100	99	100	100	100	100	100	100	100	99	100	100	100	120	110	110	100
2	A	KARUPU SWAMY	35	MALE	29972	2	82	74	72	70	72	70	70	74	78	78	80	82	82	82	82	82	82	NIL	100	100	99	100	99	100	100	99	100	100	100	100	100	100	100	100	100	110	110	110	110
3	A	MUSTAFA	32	MALE	31057	1	82	80	80	80	80	82	80	84	84	84	84	84	84	84	84	84	84	NIL	99	100	99	100	100	99	100	100	100	100	100	100	100	100	100	100	100	110	110	110	100
4	A	SANKAR	26	MALE	26417	1	78	74	72	72	72	74	74	74	72	72	72	74	76	76	78	78	78	NIL	100	100	99	100	99	100	100	100	100	100	99	100	100	100	100	100	100	110	100	100	100
5	A	MURUGAN	34	MALE	23057	1	86	84	82	70	68	72	74	72	72	70	68	70	68	70	70	72	70	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	99	110	100	100	90
6	A	VELLATHAI	56	FEMALE	59311	2	72	69	69	69	57	63	60	64	60	59	68	64	64	68	68	72	72	YES	99	99	98	98	100	100	100	100	100	100	100	100	100	100	100	100	100	140	130	120	120
7	A	SHANMUGAVEL	55	MALE	32488	2	78	76	70	70	70	72	74	76	72	68	76	80	80	82	82	82	84	NIL	99	99	98	98	100	98	100	100	100	100	100	100	100	100	100	100	100	110	110	100	100
8	A	ARUMUGHAM	54	MALE	31057	2	74	76	74	72	72	78	76	72	72	72	72	70	78	80	80	82	82	NIL	100	100	99	100	99	99	100	100	100	100	100	100	100	100	100	100	100	130	120	110	100
9	A	MANI	58	MALE	33694	1	72	68	68	72	74	72	72	68	68	70	70	70	70	70	70	72	72	NIL	100	100	99	100	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	90	90
10	A	THOMAS	35	MALE	34833	1	82	80	78	76	80	84	86	80	78	78	76	76	80	80	82	84	88	NIL	100	100	99	100	100	100	100	100	100	100	100	100	100	100	99	100	100	120	110	110	100
11	A	PARVATHI	42	FEMALE	4329	2	82	74	72	70	72	70	70	74	78	78	80	82	82	82	82	82	82	NIL	100	99	98	98	99	100	100	100	100	100	100	100	100	100	100	100	100	110	110	110	110
12	A	ALLITHURAI	26	MALE	34849	1	82	80	80	80	80	82	80	84	84	84	84	84	84	84	84	84	84	NIL	100	100	99	99	99	99	100	100	100	100	100	100	100	100	100	100	100	110	110	110	100
13	A	LEKSHMI	52	FEMALE	32592	2	78	74	72	72	72	74	74	74	72	72	72	74	76	76	78	78	78	NIL	100	100	99	100	100	99	100	99	100	100	100	100	100	100	100	100	100	110	100	100	100
14	A	ANPALAGAN	35	MALE	34866	1	86	84	82	70	68	72	74	72	72	70	68	70	68	70	70	72	70	NIL	100	99	98	98	99	100	100	100	99	100	100	100	100	100	100	100	100	110	100	100	90
15	A	SIVAKAMI	46	FEMALE	39054	1	72	69	69	69	57	63	60	64	60	59	68	64	64	68	68	72	72	NIL	100	100	99	100	100	100	99	100	100	100	100	99	100	100	100	100	100	140	130	120	120
16	A	SITALEKSHMI	45	FEMALE	41182	2	78	76	70	70	70	72	74	76	72	68	76	80	80	82	82	82	84	NIL	100	100	99	99	100	100	100	100	100	100	100	100	100	100	100	100	100	110	110	100	100
17	A	MUPPIDATI	42	FEMALE	42110	1	74	76	74	72	72	78	76	72	72	72	72	70	78	80	80	82	82	NIL	99	100	99	99	100	100	100	100	100	100	100	100	100	100	100	100	100	130	120	110	100
18	A	KASINADAN	50	MALE	33752	2	72	68	68	72	74	72	72	68	68	70	70	70	70	70	70	72	72	NIL	100	99	99	99	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	90	90
19	A	SHANMUAVADIVU	56	FEMALE	33753	2	82	80	78	76	80	84	86	80	78	78	76	76	80	80	82	84	88	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	120	120	120
20	A	ULAGARANI	49	FEMALE	38702	2	82	74	72	70	72	70	70	74	78	78	80	82	82	82	82	82	82	NIL	100	100	99	100	100	100	100	99	100	100	100	100	100	100	100	100	100	130	130	120	100

MASTER CHART: GROUP 'B' - BUPIVACAINE WITH 10µg DEXMEDETOMIDINE

							HR																		SPO2																		SBP			
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	ATR OPINE	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN
21	B	RAMASWAMY	39	MALE	33499	1	80	90	90	90	80	80	90	70	80	84	84	84	76	76	76	78	80	NIL	99	100	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	100	100	100
22	B	MARIAMMAL	35	FEMALE	37928	2	90	88	86	86	86	80	80	80	82	82	82	82	84	84	84	84	84	NIL	100	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	120	120	110	110
23	B	LEKSHMI	50	FEMALE	39018	2	80	82	84	88	84	86	82	88	86	84	82	88	82	84	88	88	88	NIL	100	99	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	108	110	110
24	B	MARIAPPAN	40	MALE	38700	1	88	84	80	78	80	82	84	88	84	84	82	80	80	78	78	84	84	NIL	99	100	99	98	100	100	100	100	100	100	100	100	100	100	100	100	100	120	110	110	100
25	B	BHAGAVATHI	48	FEMALE	4408	1	92	88	86	90	84	82	90	88	88	90	92	84	86	88	84	84	84	NIL	100	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	110	110	110
26	B	SARASWATI	50	FEMALE	4431	2	78	74	68	58	58	60	58	64	68	72	74	74	72	74	74	76	76	YES	100	99	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	110	110	110	100
27	B	ARULRAJ	48	MALE	34715	1	102	100	94	94	90	88	86	90	88	92	92	94	94	92	92	90	90	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	100	100	100
28	B	PANDIRAJAN	39	MALE	34623	1	108	112	114	108	106	104	106	98	98	96	94	98	100	102	102	104	104	NIL	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	100	100	90
29	B	KALAISELVI	46	FEMALE	5857	1	78	74	76	72	70	73	78	78	78	80	82	78	74	68	68	68	70	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	140	130	120	120
30	B	KAMARAJ	48	MALE	33295	1	98	96	98	94	94	92	92	90	94	90	90	90	90	88	88	88	86	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	110	100	100
31	B	AMIRTHARANI	45	FEMALE	41163	2	112	108	106	108	108	98	98	96	96	94	92	92	94	94	92	92	94	NIL	99	100	99	98	100	100	100	100	100	100	100	100	100	100	100	100	100	130	120	110	100
32	B	MUTHUKARUPPAN	54	MALE	33210	1	88	86	84	82	82	80	78	78	76	76	78	80	80	80	82	82	82	NIL	100	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	90	90
33	B	MURUGAN	55	MALE	31529	2	98	96	90	90	88	88	86	86	88	86	86	80	84	86	86	84	84	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	120	110	110	100
34	B	MANONMANI	42	FEMALE	42023	1	84	80	78	74	70	68	68	70	72	72	74	70	70	78	78	76	76	NIL	100	100	99	98	100	100	100	100	100	100	100	100	99	100	100	100	100	110	110	110	110
35	B	ABDUL KHASIM	51	MALE	32714	1	72	68	68	70	68	70	74	72	72	70	70	74	74	78	74	74	72	NIL	100	100	100	100	100	100	100	100	99	100	100	100	100	100	100	100	100	110	110	110	100
36	B	KUMAR	38	MALE	31240	1	96	94	92	92	92	98	100	102	108	100	98	98	96	96	98	98	96	NIL	100	100	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	110	100	100	100
37	B	SANTHOSAM	45	FEMALE	39003	1	84	80	78	76	74	76	74	72	72	70	76	76	74	74	72	72	72	NIL	100	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	100	100	90
38	B	KANMANI	49	FEMALE	36928	2	84	80	78	70	68	68	70	64	64	68	68	68	68	68	66	68	68	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	140	130	120	120
39	B	RAJAMANI	52	MALE	31285	1	78	74	70	72	78	78	76	80	82	84	86	88	88	86	86	86	88	NIL	100	99	100	99	100	100	100	100	99	100	100	100	100	100	99	100	100	110	110	100	100
40	B	PANDIAMMAL	46	FEMALE	5383	1	90	88	86	86	88	86	84	84	82	82	80	80	80	84	84	84	82	NIL	100	99	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	120	110	100

MASTER CHART: GROUP 'C' - BUPIVACAINE WITH 15µg DEXMEDETOMIDINE

							HR																			SPO2																				SBP			
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	ATR OPINE	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN
41	C	MUTHUMALAI	50	FEMALE	4401	2	92	82	86	92	82	86	90	90	88	92	95	92	88	84	84	84	88	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	120	110	100
42	C	PAVANASAM	33	MALE	51238	1	77	76	74	69	68	69	70	72	74	74	70	72	72	72	72	72	74	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	110	110	106
43	C	RAJAMMAL	45	FEMALE	56546	1	75	68	70	67	64	63	66	64	69	58	58	64	64	70	70	70	72	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	149	130	108	103
44	C	MARIAMMAL	45	FEMALE	55539	1	120	116	98	98	96	90	80	80	70	74	76	90	88	84	80	80	80	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	120	110	110	100
45	C	SUMSUDIN	49	MALE	47549	2	85	70	70	70	65	65	60	70	60	68	70	71	70	70	72	72	72	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	150	160	120	120
46	C	RAMACHANDRAN	19	MALE	50397	2	92	96	94	94	92	88	75	75	62	71	78	74	74	72	72	74	74	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	87	123	106
47	C	VELSAMY	43	MALE	53937	1	91	78	73	84	73	61	64	68	60	64	68	72	68	74	68	68	72	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	155	155	147	146
48	C	MARIYAL	42	FEMALE	41441	2	74	68	58	58	60	58	60	62	64	68	68	68	64	68	68	74	74	YES	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	120	100	100	90
49	C	PERUMAL	48	MALE	39055	1	102	100	100	90	90	90	102	98	80	86	88	88	90	90	100	98	98	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	110	120	110
50	C	JOTHI	35	FEMALE	33509	1	80	78	70	60	70	70	70	68	68	70	68	68	68	70	68	68	68	NIL	100	98	98	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	100	110	98
51	C	ESAKI MUTHU	45	MALE	39669	1	84	90	92	82	90	84	90	82	82	80	80	82	86	84	88	86	84	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	110	110	110
52	C	MANI	49	MALE	39601	1	80	78	74	72	70	68	70	68	72	76	72	74	70	76	70	70	72	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	110	110	110
53	C	PITCHAMMAL	45	FEMALE	38118	2	78	76	76	74	74	76	78	76	72	72	70	70	70	72	72	72	74	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	130	110	90	86
54	C	LEKSHMIAMMAL	55	FEMALE	40195	2	78	74	68	60	58	58	60	54	60	62	64	62	62	62	64	64	64	YES	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	150	160	120	120
55	C	CHELAPPAN	46	MALE	34201	1	106	106	104	102	100	98	102	102	104	104	104	106	100	100	100	100	102	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	87	123	106
56	C	SULAIMAN	40	MALE	324102	1	92	88	86	86	84	88	86	84	84	86	88	88	88	88	88	86	88	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	155	155	147	146
57	C	GANDHIMATHI	50	FEMALE	37142	1	84	82	78	78	76	76	78	80	82	84	86	88	86	88	84	88	86	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	120	110	90	90
58	C	MANIAMMAL	46	FEMALE	39140	2	68	74	68	72	78	80	82	84	88	82	84	84	82	82	84	84	84	NIL	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	110	110	110	106
59	C	MUTHIAH	49	MALE	30119	1	84	82	82	78	82	84	86	88	90	92	94	94	96	98	100	102	102	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	149	130	108	103
60	C	PALAMMAL	54	FEMALE	39141		78	76	74	74	72	78	78	78	74	76	78	80	80	80	80	80	84	NIL	100	99	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	120	110	110	100

MASTER CHART: GROUP 'A' - BUPIVACAINE WITH 5µg DEXMEDETOMIDINE

							SBP													DBP																MEAN BP					
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN
1	A	ABDUL KHADAR	59	MALE	35988	2	100	100	90	100	100	100	100	100	100	100	100	100	100	80	70	70	70	70	68	72	74	76	78	78	80	80	82	82	82	82	90	88	86	84	84	84
2	A	KARUPU SWAMY	35	MALE	29972	2	120	110	120	130	120	120	120	120	110	110	110	100	100	66	66	66	68	68	68	68	60	70	70	70	74	74	74	74	74	74	90	90	90	90	90	90
3	A	MUSTAFA	32	MALE	31057	1	100	100	100	100	110	110	110	110	110	110	110	110	110	70	72	72	70	70	74	70	72	70	72	74	74	74	74	74	74	74	82	82	82	82	82	82
4	A	SANKAR	26	MALE	26417	1	100	100	100	100	110	110	110	110	110	110	110	110	110	80	70	72	70	72	70	70	70	72	80	80	80	80	80	80	80	80	90	80	80	80	80	80
5	A	MURUGAN	34	MALE	23057	1	80	100	80	100	100	100	100	100	100	100	100	100	100	90	80	80	70	64	70	64	70	70	70	70	70	70	70	70	70	70	90	80	80	70	64	70
6	A	VELLATHAI	56	FEMALE	59311	2	120	130	130	140	140	150	150	120	120	130	130	130	130	80	80	80	80	80	80	80	80	90	80	80	80	80	80	80	80	80	100	96	92	92	92	96
7	A	SHANMUGAVEL	55	MALE	32488	2	100	100	90	90	110	110	110	100	100	100	100	100	100	80	80	70	70	70	70	60	60	70	80	80	70	70	70	70	70	70	90	90	80	80	80	80
8	A	ARUMUGHAM	54	MALE	31057	2	100	100	100	110	110	110	110	120	120	120	120	120	120	90	80	80	60	60	60	70	70	70	70	70	80	80	80	80	80	80	103	94	73	73	73	80
9	A	MANI	58	MALE	33694	1	100	100	96	96	100	100	100	100	100	106	106	106	110	70	70	60	60	60	60	60	60	66	66	70	70	70	66	66	68	68	80	80	70	70	73	73
10	A	THOMAS	35	MALE	34833	1	100	100	90	100	100	100	100	100	100	100	100	100	100	80	70	70	70	70	68	72	74	76	78	78	80	80	82	82	82	82	90	88	86	84	84	84
11	A	PARVATHI	42	FEMALE	4329	2	120	110	120	130	120	120	120	120	110	110	110	100	100	66	66	66	68	68	68	68	60	70	70	70	74	74	74	74	74	74	90	90	90	90	90	90
12	A	ALLITHURAI	26	MALE	34849	1	100	100	100	100	110	110	110	110	110	110	110	110	110	70	72	72	70	70	74	70	72	70	72	74	74	74	74	74	74	74	82	82	82	82	82	82
13	A	LEKSHMI	52	FEMALE	32592	2	100	100	100	100	110	110	110	110	110	110	110	110	110	80	70	72	70	72	70	70	70	72	80	80	80	80	80	80	80	80	90	80	80	80	80	80
14	A	ANPALAGAN	35	MALE	34866	1	80	100	80	100	100	100	100	100	100	100	100	100	100	90	80	80	70	64	70	64	70	70	70	70	70	70	70	70	70	70	90	80	80	70	64	70
15	A	SIVAKAMI	46	FEMALE	39054	1	120	130	130	140	140	150	150	120	120	130	130	130	130	80	80	80	80	80	80	80	80	90	80	80	80	80	80	80	80	80	100	96	92	92	92	96
16	A	SITALEKSHMI	45	FEMALE	41182	2	100	100	90	90	110	110	110	100	100	100	100	100	100	80	80	70	70	70	70	60	60	70	80	80	70	70	70	70	70	70	90	90	80	80	80	80
17	A	MUPPIDATI	42	FEMALE	42110	1	100	100	100	110	110	110	110	120	120	120	120	120	120	90	80	80	60	60	60	70	70	70	70	70	80	80	80	80	80	80	103	94	73	73	73	80
18	A	KASINADAN	50	MALE	33752	2	100	100	96	96	100	100	100	100	100	106	106	106	110	70	70	60	60	60	60	60	60	66	66	70	70	70	66	66	68	68	80	80	70	70	73	73
19	A	SHANMUAVADIVU	56	FEMALE	33753	2	110	110	110	110	110	110	110	110	110	110	110	110	110	80	80	80	80	70	70	70	70	70	70	70	70	70	70	70	70	70	96	93	93	93	83	83
20	A	ULAGARANI	49	FEMALE	38702	2	100	90	90	100	100	96	96	100	100	100	100	106	106	80	80	80	70	70	60	60	60	60	60	60	66	66	70	70	66	66	96	96	93	80	80	70

MASTER CHART: GROUP 'B' - BUPIVACAINE WITH 10µg DEXMEDETOMIDINE

							SBP													DBP															MEAN BP					
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN
21	B	RAMASWAMY	39	MALE	33499	1	100	100	100	110	110	110	120	120	120	120	120	120	120	80	80	80	70	60	50	50	60	60	60	70	70	70	70	70	70	70	70	68	70	64	68	70
22	B	MARIAMMAL	35	FEMALE	37928	2	110	106	106	110	108	108	100	110	100	100	100	100	100	80	80	80	80	80	70	70	70	66	60	73	73	83	79	78	78	78	93	93	91	90	91	82
23	B	LEKSHMI	50	FEMALE	39018	2	100	110	110	110	108	108	110	110	110	108	110	120	120	70	70	52	52	62	70	70	70	70	70	68	68	68	68	70	70	72	82	80	74	74	77	90
24	B	MARIAPPAN	40	MALE	38700	1	100	100	90	100	100	100	100	100	100	100	100	100	100	80	70	70	70	70	68	72	74	76	78	78	80	80	82	82	82	82	90	88	86	84	84	84
25	B	BHAGAVATHI	48	FEMALE	4408	1	120	110	120	130	120	120	120	120	110	110	110	100	100	66	66	66	68	68	68	68	60	70	70	70	74	74	74	74	74	74	90	90	90	90	90	90
26	B	SARASWATI	50	FEMALE	4431	2	100	100	100	100	110	110	110	110	110	110	110	110	110	70	72	72	70	70	74	70	72	70	72	74	74	74	74	74	74	74	82	82	82	82	82	82
27	B	ARULRAJ	48	MALE	34715	1	100	100	100	100	110	110	110	110	110	110	110	110	110	80	70	72	70	72	70	70	70	72	80	80	80	80	80	80	80	80	90	80	80	80	80	80
28	B	PANDIRAJAN	39	MALE	34623	1	80	100	80	100	100	100	100	100	100	100	100	100	100	90	80	80	70	64	70	64	70	70	70	70	70	70	70	70	70	70	90	80	80	70	64	70
29	B	KALAISELVI	46	FEMALE	5857	1	120	130	130	140	140	150	150	120	120	130	130	130	130	80	80	80	80	80	80	80	80	90	80	80	80	80	80	80	80	80	100	96	92	92	92	96
30	B	KAMARAJ	48	MALE	33295	1	100	100	90	90	110	110	110	100	100	100	100	100	100	80	80	70	70	70	70	60	60	70	80	80	70	70	70	70	70	70	90	90	80	80	80	80
31	B	AMIRTHARANI	45	FEMALE	41163	2	100	100	100	110	110	110	110	120	120	120	120	120	120	90	80	80	60	60	60	70	70	70	70	70	80	80	80	80	80	80	103	94	73	73	73	80
32	B	MUTHUKARUPPAN	54	MALE	33210	1	100	100	96	96	100	100	100	100	100	106	106	106	110	70	70	60	60	60	60	60	60	66	66	70	70	70	66	66	68	68	80	80	70	70	73	73
33	B	MURUGAN	55	MALE	31529	2	100	100	90	100	100	100	100	100	100	100	100	100	100	80	70	70	70	70	68	72	74	76	78	78	80	80	82	82	82	82	90	88	86	84	84	84
34	B	MANONMANI	42	FEMALE	42023	1	120	110	120	130	120	120	120	120	110	110	110	100	100	66	66	66	68	68	68	68	60	70	70	70	74	74	74	74	74	74	90	90	90	90	90	90
35	B	ABDUL KHASIM	51	MALE	32714	1	100	100	100	100	110	110	110	110	110	110	110	110	110	70	72	72	70	70	74	70	72	70	72	74	74	74	74	74	74	74	82	82	82	82	82	82
36	B	KUMAR	38	MALE	31240	1	100	100	100	100	110	110	110	110	110	110	110	110	110	80	70	72	70	72	70	70	70	72	80	80	80	80	80	80	80	80	90	80	80	80	80	80
37	B	SANTHOSAM	45	FEMALE	39003	1	80	100	80	100	100	100	100	100	100	100	100	100	100	90	80	80	70	64	70	64	70	70	70	70	70	70	70	70	70	70	90	80	80	70	64	70
38	B	KANMANI	49	FEMALE	36928	2	120	130	130	140	140	150	150	120	120	130	130	130	130	80	80	80	80	80	80	80	80	90	80	80	80	80	80	80	80	80	100	96	92	92	92	96
39	B	RAJAMANI	52	MALE	31285	1	100	100	90	90	110	110	110	100	100	100	100	100	100	80	80	70	70	70	70	60	60	70	80	80	70	70	70	70	70	70	90	90	80	80	80	80
40	B	PANDIAMMAL	46	FEMALE	5383	1	100	100	100	110	110	110	110	120	120	120	120	120	120	90	80	80	60	60	60	70	70	70	70	70	80	80	80	80	80	80	103	94	73	73	73	80

MASTER CHART: GROUP 'C' - BUPIVACAINE WITH 15µg DEXMEDETOMIDINE

							SBP																		DBP														MEAN BP					
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN
41	C	MUTHUMALAI	50	FEMALE	4401	2	100	100	100	110	110	110	110	120	120	120	120	120	120	90	80	80	60	60	60	70	70	70	70	70	80	80	80	80	80	80	103	94	73	73	73	80
42	C	PAVANASAM	33	MALE	51238	1	110	100	100	100	10	110	110	100	100	100	100	100	100	70	70	60	60	60	60	60	60	60	60	60	60	60	60	70	70	83	83	78	77	78	78
43	C	RAJAMMAL	45	FEMALE	56546	1	100	128	118	113	120	103	100	100	100	100	110	110	110	88	72	58	53	51	76	63	57	63	53	70	70	70	70	70	70	70	110	92	78	70	72	99
44	C	MARIAMMAL	45	FEMALE	55539	1	100	120	110	110	110	100	110	110	100	110	110	110	110	80	80	80	80	90	90	90	80	80	80	80	80	70	80	70	70	70	93	90	90	86	93	100
45	C	SUMSUDIN	49	MALE	47549	2	120	120	130	120	118	138	130	120	120	110	120	120	130	90	89	74	75	70	70	75	75	75	71	70	72	70	68	70	75	80	106	105	101	93	92	92
46	C	RAMACHANDRAN	19	MALE	50397	2	98	100	102	105	101	111	151	110	110	110	110	110	110	67	55	64	57	58	59	54	55	52	71	85	70	70	70	67	67	70	81	69	82	77	75	87
47	C	VELSAMY	43	MALE	53937	1	141	153	117	139	127	117	100	100	117	110	110	100	110	87	87	92	83	75	119	86	70	49	86	70	70	86	70	70	70	70	112	112	116	106	108	139
48	C	MARIYAL	42	FEMALE	41441	2	90	100	100	100	90	110	110	100	100	110	110	110	110	80	70	70	60	60	70	70	70	60	72	72	70	70	72	72	72	72	93	80	80	70	70	80
49	C	PERUMAL	48	MALE	39055	1	110	110	98	90	88	90	90	90	100	100	100	100	100	80	70	70	60	60	60	60	60	60	60	60	68	68	68	68	60	60	80	80	80	80	80	74
50	C	JOTHI	35	FEMALE	33509	1	110	120	110	100	100	100	100	104	104	110	110	110	110	80	80	70	60	70	70	70	70	72	72	68	68	68	68	70	70	70	80	83	73	83	93	85
51	C	ESAKI MUTHU	45	MALE	39669	1	100	100	100	102	110	110	110	108	108	120	120	120	120	80	72	70	70	70	70	70	72	72	80	80	80	80	80	80	80	80	96	90	86	83	80	80
52	C	MANI	49	MALE	39601	1	100	100	100	100	110	110	110	110	110	110	120	120	120	80	80	74	70	70	70	70	70	70	70	72	70	80	80	80	82	80	96	90	86	83	80	80
53	C	PITCHAMMAL	45	FEMALE	38118	2	90	100	100	100	110	110	110	110	110	110	120	120	120	80	80	60	55	60	70	70	70	70	70	72	70	80	80	80	82	80	96	90	70	65	70	80
54	C	LEKSHMIAMMAL	55	FEMALE	40195	2	120	120	130	120	118	138	130	120	120	110	120	120	130	90	89	74	75	70	70	75	75	75	71	70	72	70	68	70	75	80	106	105	101	93	92	92
55	C	CHELAPPAN	46	MALE	34201	1	98	100	102	105	101	111	151	110	110	110	110	110	110	67	55	64	57	58	59	54	55	52	71	85	70	70	70	67	67	70	81	69	82	77	75	87
56	C	SULAIMAN	40	MALE	324102	1	141	153	117	139	127	117	100	100	117	110	110	100	110	87	87	92	83	75	119	86	70	49	86	70	70	86	70	70	70	70	112	112	116	106	108	139
57	C	GANDHIMATHI	50	FEMALE	37142	1	88	90	100	100	100	100	110	110	110	100	100	100	100	80	80	60	60	60	60	80	80	80	80	70	70	70	70	70	70	70	93	90	70	70	69	70
58	C	MANIAMMAL	46	FEMALE	39140	2	110	100	100	100	10	110	110	100	100	100	100	100	100	70	70	60	60	60	60	60	60	60	60	60	60	60	60	60	70	70	83	83	78	77	78	78
59	C	MUTHIAH	49	MALE	30119	1	100	128	118	113	120	103	100	100	100	100	110	110	110	88	72	58	53	51	76	63	57	63	53	70	70	70	70	70	70	70	110	92	78	70	72	99
60	C	PALAMMAL	54	FEMALE	39141		100	120	110	110	110	100	110	110	100	110	110	110	110	80	80	80	80	90	90	90	80	80	80	80	80	70	80	70	70	70	93	90	90	86	93	100

MASTER CHART: GROUP 'A' - BUPIVACAINE WITH 5µg DEXMEDETOMIDINE

							MEAN BP												RR																											
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	VAS OPR ESS OR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	OSB (SEC)	OMB (SEC)	MSL	MAX MOTOR BLOCK	TIME TO TWO SEGMENT REGRESSION (MIN)	DURATION OF SURGERY (MIN)
1	A	ABDUL KHADAR	59	MALE	35988	2	84	84	88	88	88	88	84	84	88	88	88	NIL	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	250	180	T8	3	180	65
2	A	KARUPU SWAMY	35	MALE	29972	2	90	98	96	98	98	94	96	98	92	98	96	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	240	230	T6	3	105	75
3	A	MUSTAFA	32	MALE	31057	1	80	80	80	80	80	80	80	80	80	80	80	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	220	240	T8	3	110	60
4	A	SANKAR	26	MALE	26417	1	80	80	80	90	90	90	90	90	90	90	90	NIL	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	200	200	T8	3	125	68
5	A	MURUGAN	34	MALE	23057	1	64	70	70	70	70	70	70	70	70	70	72	YES	18	20	20	20	20	18	18	18	16	18	18	18	18	20	20	20	20	220	210	T8	3	130	75
6	A	VELLATHAI	56	FEMALE	59311	2	96	100	106	93	93	92	92	96	96	96	96	NIL	16	16	16	16	16	18	18	18	18	20	20	20	20	20	20	20	18	185	250	T8	3	180	180
7	A	SHANMUGAVEL	55	MALE	32488	2	70	70	83	90	90	83	83	83	80	80	80	NIL	18	18	18	18	18	20	20	20	20	18	18	18	20	20	20	22	22	195	255	T8	3	300	90
8	A	ARUMUGHAM	54	MALE	31057	2	80	80	80	80	94	94	94	94	94	94	94	NIL	20	20	20	20	22	22	22	24	24	22	18	18	18	20	20	20	20	210	270	T6	3	260	80
9	A	MANI	58	MALE	33694	1	72	72	77	77	80	80	80	79	79	80	80	NIL	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	200	230	T7	3	110	68
10	A	THOMAS	35	MALE	34833	1	84	84	88	88	88	88	84	84	88	88	88	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	190	240	T8	3	140	75
11	A	PARVATHI	42	FEMALE	4329	2	90	98	96	98	98	94	96	98	92	98	96	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	220	200	T8	3	120	130
12	A	ALLITHURAI	26	MALE	34849	1	80	80	80	80	80	80	80	80	80	80	80	NIL	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	210	210	T8	3	110	80
13	A	LEKSHMI	52	FEMALE	32592	2	80	80	80	90	90	90	90	90	90	90	90	NIL	18	20	20	20	20	18	18	18	16	18	18	18	18	20	20	20	20	240	245	T6	3	125	140
14	A	ANPALAGAN	35	MALE	34866	1	64	70	70	70	70	70	70	70	70	70	72	NIL	16	16	16	16	16	18	18	18	18	20	20	20	20	20	20	20	18	280	260	T6	3	130	65
15	A	SIVAKAMI	46	FEMALE	39054	1	96	100	106	93	93	92	92	96	96	96	96	NIL	18	18	18	18	18	20	20	20	20	18	18	18	20	20	20	22	22	290	250	T6	3	180	140
16	A	SITALEKSHMI	45	FEMALE	41182	2	70	70	83	90	90	83	83	83	80	80	80	NIL	20	20	20	20	22	22	22	24	24	22	18	18	18	20	20	20	20	246	260	T8	3	150	120
17	A	MUPPIDATI	42	FEMALE	42110	1	80	80	80	80	94	94	94	94	94	94	94	NIL	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	230	240	T8	3	110	136
18	A	KASINADAN	50	MALE	33752	2	72	72	77	77	80	80	80	79	79	80	80	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	260	230	T8	3	200	75
19	A	SHANMUAVADIVU	56	FEMALE	33753	2	83	83	83	83	83	83	83	83	83	83	83	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	210	235	T8	3	210	120
20	A	ULAGARANI	49	FEMALE	38702	2	70	73	73	72	72	77	77	80	80	79	79	NIL	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	225	225	T7	3	130	110

MASTER CHART: GROUP 'B' - BUPIVACAINE WITH 10µg DEXMEDETOMIDINE

							MEAN BP												RR																								
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	VAS OPRESSOR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	OSB (SEC)	OMB (SEC)	MSL	MAX MOTOR BLOCK	TIME TO TWO SEGMENT REGRESSION (MIN)	DURATION OF SURGERY (MIN)
21	B	RAMASWAMY	39	MALE	33499	1	62	77	82	75	86	86	86	86	86	86	86	YES	18	20	20	20	20	18	18	18	16	18	18	18	18	20	20	20	20	180	180	T6	3	145	75
22	B	MARIAMMAL	35	FEMALE	37928	2	82	83	80	76	83	85	88	85	86	86	86	NIL	16	16	16	16	16	18	18	18	18	20	20	20	20	20	20	20	18	240	220	T6	3	130	160
23	B	LEKSHMI	50	FEMALE	39018	2	84	94	90	85	85	82	86	84	88	90	91	NIL	18	18	18	18	18	20	20	20	20	18	18	18	20	20	20	22	22	200	225	T8	3	110	120
24	B	MARIAPPAN	40	MALE	38700	1	84	84	88	88	88	88	84	84	88	88	88	NIL	20	20	20	20	22	22	22	24	24	22	18	18	18	20	20	20	20	185	215	T7	3	140	68
25	B	BHAGAVATHI	48	FEMALE	4408	1	90	98	96	98	98	94	96	98	92	98	96	NIL	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	200	240	T7	3	135	110
26	B	SARASWATI	50	FEMALE	4431	2	80	80	80	80	80	80	80	80	80	80	80	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	240	245	T7	3	160	140
27	B	ARULRAJ	48	MALE	34715	1	80	80	80	90	90	90	90	90	90	90	90	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	250	250	T8	3	180	90
28	B	PANDIRAJAN	39	MALE	34623	1	64	70	70	70	70	70	70	70	70	70	72	YES	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	190	220	T8	3	160	80
29	B	KALAISELVI	46	FEMALE	5857	1	96	100	106	93	93	92	92	96	96	96	96	NIL	18	20	20	20	20	18	18	18	16	18	18	18	18	20	20	20	20	210	230	T8	3	145	136
30	B	KAMARAJ	48	MALE	33295	1	70	70	83	90	90	83	83	83	80	80	80	NIL	16	16	16	16	16	18	18	18	18	20	20	20	20	20	20	20	18	220	210	T4	3	200	65
31	B	AMIRTHARANI	45	FEMALE	41163	2	80	80	80	80	94	94	94	94	94	94	94	NIL	18	18	18	18	18	20	20	20	20	18	18	18	20	20	20	22	22	195	220	T6	3	120	120
32	B	MUTHUKARUPPAN	54	MALE	33210	1	72	72	77	77	80	80	80	79	79	80	80	NIL	20	20	20	20	22	22	22	24	24	22	18	18	18	20	20	20	20	200	240	T6	3	124	75
33	B	MURUGAN	55	MALE	31529	2	84	84	88	88	88	88	84	84	88	88	88	NIL	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	190	210	T8	3	175	80
34	B	MANONMANI	42	FEMALE	42023	1	90	98	96	98	98	94	96	98	92	98	96	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	210	230	T7	2	280	110
35	B	ABDUL KHASIM	51	MALE	32714	1	80	80	80	80	80	80	80	80	80	80	80	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	180	240	T7	3	200	65
36	B	KUMAR	38	MALE	31240	1	80	80	80	90	90	90	90	90	90	90	90	NIL	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	210	245	T6	3	120	70
37	B	SANTHOSAM	45	FEMALE	39003	1	64	70	70	70	70	70	70	70	70	70	72	NIL	18	20	20	20	20	18	18	18	16	18	18	18	18	20	20	20	20	230	250	T8	3	110	126
38	B	KANMANI	49	FEMALE	36928	2	96	100	106	93	93	92	92	96	96	96	96	NIL	16	16	16	16	16	18	18	18	18	20	20	20	20	20	20	20	18	200	235	T8	3	114	130
39	B	RAJAMANI	52	MALE	31285	1	70	70	83	90	90	83	83	83	80	80	80	NIL	18	18	18	18	18	20	20	20	20	18	18	18	20	20	20	22	22	195	230	T7	3	126	68
40	B	PANDIAMMAL	46	FEMALE	5383	1	80	80	80	80	94	94	94	94	94	94	94	NIL	20	20	20	20	22	22	22	24	24	22	18	18	18	20	20	20	20	210	230	T8	3	110	130

MASTER CHART: GROUP 'C' - BUPIVACAINE WITH 15µg DEXMEDETOMIDINE

							MEAN BP												RR																						
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	VASOPRESSOR	0 MIN	5 MIN	10 MIN	15 MIN	20 MIN	30 MIN	45 MIN	60 MIN	90 MIN	120 MIN	3 HR	4 HR	8 HR	12 HR	16 HR	20 HR	24 HR	OSB (SEC)	OMB (SEC)	MSL	MAX MOTOR BLOCK	TIME TO TWO SEGMENT REGRESSION (MIN)	DURATION OF SURGERY (MIN)
41	C	MUTHUMALAI	50	FEMALE	4401	2	80	80	80	80	94	94	94	94	94	94	94	YES	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	200	215	T4	3	190	90
42	C	PAVANASAM	33	MALE	51238	1	70	70	78	78	78	70	70	70	70	80	80	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	190	206	T6	3	180	90
43	C	RAJAMMAL	45	FEMALE	56546	1	84	79	82	70	80	80	80	80	83	83	83	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	185	195	T4	3	200	110
44	C	MARIAMMAL	45	FEMALE	55539	1	96	90	90	86	90	90	80	90	84	84	84	NIL	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	188	185	T7	3	180	90
45	C	SUMSUDIN	49	MALE	47549	2	96	88	89	105	90	88	92	82	92	88	96	NIL	18	20	20	20	20	18	18	18	16	18	18	18	18	20	20	20	20	195	190	T6	3	165	150
46	C	RAMACHANDRAN	19	MALE	50397	2	73	74	70	84	114	84	84	84	81	81	84	YES	16	16	16	16	16	18	18	18	18	20	20	20	20	20	20	20	18	210	185	T6	3	200	140
47	C	VELSAMY	43	MALE	53937	1	104	94	85	104	80	80	104	84	84	80	84	NIL	18	18	18	18	18	20	20	20	20	18	18	18	20	20	20	22	22	220	190	T4	3	240	90
48	C	MARIYAL	42	FEMALE	41441	2	80	80	70	84	84	80	80	84	84	84	84	NIL	20	20	20	20	22	22	22	24	24	22	18	18	18	20	20	20	20	180	195	T4	3	120	105
49	C	PERUMAL	48	MALE	39055	1	74	75	75	76	76	74	74	72	72	70	69	NIL	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	195	220	T7	3	140	130
50	C	JOTHI	35	FEMALE	33509	1	73	76	79	78	79	80	83	86	83	82	78	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	200	180	T6	3	146	146
51	C	ESAKI MUTHU	45	MALE	39669	1	80	80	82	83	82	84	80	82	82	80	80	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	195	195	T6	3	130	70
52	C	MANI	49	MALE	39601	1	80	80	83	83	84	83	90	90	93	94	93	NIL	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	210	196	T4	3	150	75
53	C	PITCHAMMAL	45	FEMALE	38118	2	80	80	83	83	84	83	83	90	93	94	93	YES	18	20	20	20	20	18	18	18	16	18	18	18	18	20	20	20	20	185	210	T4	3	180	110
54	C	LEKSHMIAMMAL	55	FEMALE	40195	2	96	88	89	105	90	88	92	82	92	88	96	NIL	16	16	16	16	16	18	18	18	18	20	20	20	20	20	20	20	18	190	187	T6	3	135	126
55	C	CHELAPPAN	46	MALE	34201	1	73	74	70	84	114	84	84	84	81	81	84	NIL	18	18	18	18	18	20	20	20	20	18	18	18	20	20	20	22	22	210	190	T7	3	160	80
56	C	SULAIMAN	40	MALE	324102	1	104	94	85	104	80	80	104	84	84	80	84	NIL	20	20	20	20	22	22	22	24	24	22	18	18	18	20	20	20	20	240	180	T6	3	180	90
57	C	GANDHIMATHI	50	FEMALE	37142	1	86	86	86	86	83	83	83	80	80	80	80	NIL	18	16	18	20	20	18	18	18	18	20	18	18	18	20	18	18	18	195	170	T4	3	168	120
58	C	MANIAMMAL	46	FEMALE	39140	2	70	70	78	78	78	70	70	70	70	80	80	NIL	18	18	16	18	18	18	20	20	16	18	18	20	20	18	18	18	18	180	180	T4	3	200	140
59	C	MUTHIAH	49	MALE	30119	1	84	79	82	70	80	80	80	80	83	83	83	NIL	18	16	18	16	16	18	18	18	18	16	18	18	18	16	16	16	18	195	170	T8	3	180	75
60	C	PALAMMAL	54	FEMALE	39141		96	90	90	86	90	90	80	90	84	84	84	NIL	18	20	20	20	20	22	22	22	20	20	20	18	18	18	18	18	18	180	170	T7	3	210	134

MASTER CHART: GROUP 'A' - BUPIVACAINE WITH 5µg DEXMEDETOMIDINE

							SEDATION SCORE									VAS SCORE						ADVERSE EFFECTS				
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	1 HR	2 HR	3 HR	4 HR	8 HR	12 HR	24 HR	DSB (MIN)	DMB (MIN)	2 HR	3 HR	4 HR	8 HR	12 HR	TIME FOR RESCUE ANALGESIA	HYPOTENSION	BRADYCARDIA	NAUSEA AND VOMITING	URINARY RETENSION	PRURITUS
1	A	ABDUL KHADAR	59	MALE	35988	2	2	2	2	2	2	2	2	210	240	0	0	4			260	nil	nil	nil	nil	nil
2	A	KARUPU SWAMY	35	MALE	29972	2	2	2	2	2	2	2	2	200	260	0	0	2	4		480	nil	nil	nil	nil	nil
3	A	MUSTAFA	32	MALE	31057	1	1	2	2	2	2	2	2	240	280	0	2	4			250	nil	nil	nil	nil	nil
4	A	SANKAR	26	MALE	26417	1	1	2	2	2	2	2	2	190	290	0	0	4			300	nil	nil	nil	nil	nil
5	A	MURUGAN	34	MALE	23057	1	2	3	2	2	2	2	2	210	240	0	0	2	4		480	YES	nil	nil	nil	nil
6	A	VELLATHAI	56	FEMALE	59311	2	2	2	2	2	2	2	2	220	300	0	2	4			300	nil	YES	nil	nil	nil
7	A	SHANMUGAVEL	55	MALE	32488	2	2	2	2	2	2	2	2	180	310	0	0	4			260	nil	nil	nil	nil	nil
8	A	ARUMUGHAM	54	MALE	31057	2	2	2	2	2	2	2	2	240	260	0	0	2	4		480	nil	nil	nil	nil	nil
9	A	MANI	58	MALE	33694	1	1	2	2	2	2	2	2	220	280	0	2	4			250	nil	nil	nil	nil	nil
10	A	THOMAS	35	MALE	34833	1	1	2	2	2	2	2	2	210	300	0	0	4			300	nil	nil	nil	nil	nil
11	A	PARVATHI	42	FEMALE	4329	2	2	3	2	2	2	2	2	200	310	0	0	2	4		480	nil	nil	nil	nil	nil
12	A	ALLITHURAI	26	MALE	34849	1	2	2	2	2	2	2	2	220	300	0	2	4			300	nil	nil	nil	nil	nil
13	A	LEKSHMI	52	FEMALE	32592	2	2	2	2	2	2	2	2	240	310	0	0	4			260	nil	nil	nil	nil	nil
14	A	ANPALAGAN	35	MALE	34866	1	2	2	2	2	2	2	2	190	290	0	0	2	4		480	nil	nil	nil	nil	nil
15	A	SIVAKAMI	46	FEMALE	39054	1	1	2	2	2	2	2	2	320	200	0	2	4			250	nil	nil	nil	nil	nil
16	A	SITALEKSHMI	45	FEMALE	41182	2	1	2	2	2	2	2	2	300	210	0	0	4			300	nil	nil	nil	nil	nil
17	A	MUPPIDATI	42	FEMALE	42110	1	2	3	2	2	2	2	2	340	215	0	0	2	4		480	nil	nil	nil	nil	nil
18	A	KASINADAN	50	MALE	33752	2	2	2	2	2	2	2	2	290	218	0	2	4			300	nil	nil	nil	nil	nil
19	A	SHANMUAVADIVU	56	FEMALE	33753	2	2	2	2	2	2	2	2	290	200	0	0	4			260	nil	nil	nil	nil	nil
20	A	ULAGARANI	49	FEMALE	38702	2	2	2	2	2	2	2	2	310	200	0	0	2	4		480	nil	nil	nil	nil	nil

MASTER CHART: GROUP 'B' - BUPIVACAINE WITH 10µg DEXMEDETOMIDINE

							SEDATION SCORE									VAS SCORE						ADVERSE EFFECTS				
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	1 HR	2 HR	3 HR	4 HR	8 HR	12 HR	24 HR	DSB (MIN)	DMB (MIN)	2 HR	3 HR	4 HR	8 HR	12 HR	TIME FOR RESCUE ANALGESIA	HYPOTENSION	BRADYCARDIA	NAUSEA AND VOMITING	URINARY RETENSION	PRURITUS
21	B	RAMASWAMY	39	MALE	33499	1	2	2	2	2	2	2	2	210	310	0	2	2	4		480	YES	nil	nil	nil	nil
22	B	MARIAMMAL	35	FEMALE	37928	2	2	2	2	2	2	2	2	200	320	0	0	2	4		440	nil	nil	nil	nil	nil
23	B	LEKSHMI	50	FEMALE	39018	2	2	2	2	3	3	3	3	240	360	0	0	0	2	4	500	nil	nil	nil	nil	nil
24	B	MARIAPPAN	40	MALE	38700	1	2	2	2	2	2	2	2	300	300	0	2	2	4		480	nil	nil	nil	nil	nil
25	B	BHAGAVATHI	48	FEMALE	4408	1	2	2	2	2	2	2	2	340	340	0	2	2	4		480	nil	nil	nil	nil	nil
26	B	SARASWATI	50	FEMALE	4431	2	2	2	2	2	2	2	2	360	360	0	0	2	4		440	nil	YES	nil	nil	nil
27	B	ARULRAJ	48	MALE	34715	1	2	2	2	3	3	3	3	380	380	0	0	0	2	4	500	nil	nil	nil	nil	nil
28	B	PANDIRAJAN	39	MALE	34623	1	2	2	2	2	2	2	2	360	360	0	0	2	4		440	YES	nil	nil	nil	nil
29	B	KALAISELVI	46	FEMALE	5857	1	2	2	2	2	2	2	2	320	320	0	2	2	4		480	nil	nil	nil	nil	nil
30	B	KAMARAJ	48	MALE	33295	1	2	2	2	2	2	2	2	310	310	0	0	2	4		440	nil	nil	nil	nil	nil
31	B	AMIRTHARANI	45	FEMALE	41163	2	2	2	2	3	3	3	3	300	300	0	0	0	2	4	500	nil	nil	nil	nil	nil
32	B	MUTHUKARUPPAN	54	MALE	33210	1	2	2	2	2	2	2	2	290	290	0	2	2	4		480	nil	nil	nil	nil	nil
33	B	MURUGAN	55	MALE	31529	2	2	2	2	2	2	2	2	280	280	0	2	2	4		480	nil	nil	nil	nil	nil
34	B	MANONMANI	42	FEMALE	42023	1	2	2	2	2	2	2	2	300	300	0	0	2	4		440	nil	nil	nil	nil	nil
35	B	ABDUL KHASIM	51	MALE	32714	1	2	2	2	3	3	3	3	220	280	0	0	0	2	4	500	nil	nil	nil	nil	nil
36	B	KUMAR	38	MALE	31240	1	2	2	2	2	2	2	2	240	300	0	0	2	4		440	nil	nil	nil	nil	nil
37	B	SANTHOSAM	45	FEMALE	39003	1	2	2	2	2	2	2	2	190	270	0	2	2	4		480	nil	nil	nil	nil	nil
38	B	KANMANI	49	FEMALE	36928	2	2	2	2	2	2	2	2	320	340	0	0	2	4		440	nil	nil	nil	nil	nil
39	B	RAJAMANI	52	MALE	31285	1	2	2	2	3	3	3	3	300	300	0	0	0	2	4	500	nil	nil	nil	nil	nil
40	B	PANDIAMMAL	46	FEMALE	5383	1	2	2	2	2	2	2	2	340	340	0	2	2	4		480	nil	nil	nil	nil	nil

MASTER CHART: GROUP 'C' - BUPIVACAINE WITH 15µg DEXMEDETOMIDINE

							SEDATION SCORE									VAS SCORE						ADVERSE EFFECTS				
SNO	GR.	NAME	AGE	SEX	IP.NO	ASA	1 HR	2 HR	3 HR	4 HR	8 HR	12 HR	24 HR	DSB (MIN)	DMB (MIN)	2 HR	3 HR	4 HR	8 HR	12 HR	TIME FOR RESCUE ANALGESIA	HYPOTENSION	BRADYCARDIA	NAUSEA AND VOMITING	URINARY RETENSION	PRURITUS
41	C	MUTHUMALAI	50	FEMALE	4401	2	2	3	3	3	3	3	3	360	360	0	0	2	2	4	650	YES	nil	nil	nil	nil
42	C	PAVANASAM	33	MALE	51238	1	2	3	3	3	3	3	3	350	370	0	0	2	2	4	720	nil	nil	nil	nil	nil
43	C	RAJAMMAL	45	FEMALE	56546	1	2	2	3	4	4	4	4	340	350	0	0	2	2	4	800	nil	nil	nil	nil	nil
44	C	MARIAMMAL	45	FEMALE	55539	1	2	2	3	3	4	4	4	280	365	0	0	0	2	4	840	nil	nil	nil	nil	nil
45	C	SUMSUDIN	49	MALE	47549	2	2	2	3	3	3	3	3	280	370	0	0	2	2	4	680	nil	nil	nil	nil	nil
46	C	RAMACHANDRAN	19	MALE	50397	2	2	2	2	3	3	3	3	280	350	0	0	0	2	4	780	YES	nil	nil	nil	nil
47	C	VELSAMY	43	MALE	53937	1	2	2	3	3	4	4	4	280	340	0	0	0	2	2	750	nil	nil	nil	nil	nil
48	C	MARIYAL	42	FEMALE	41441	2	2	2	3	3	3	3	3	280	370	0	0	0	2	4	710	nil	YES	nil	nil	nil
49	C	PERUMAL	48	MALE	39055	1	2	2	3	4	4	4	4	340	390	0	0	0	2	2	750	nil	nil	nil	nil	nil
50	C	JOTHI	35	FEMALE	33509	1	2	3	3	4	4	4	4	340	395	0	0	0	2	2	800	nil	nil	nil	nil	nil
51	C	ESAKI MUTHU	45	MALE	39669	1	2	2	3	3	3	3	3	300	340	0	0	2	4		600	nil	nil	nil	nil	nil
52	C	MANI	49	MALE	39601	1	2	2	3	4	4	4	4	300	335	0	0	0	2	4	730	nil	nil	nil	nil	nil
53	C	PITCHAMMAL	45	FEMALE	38118	2	2	2	3	3	4	4	4	360	360	0	0	0	2	4	730	YES	nil	nil	nil	nil
54	C	LEKSHMIAMMAL	55	FEMALE	40195	2	2	2	3	3	3	3	3	380	360	0	0	0	2	2	800	nil	YES	nil	nil	nil
55	C	CHELAPPAN	46	MALE	34201	1	2	3	3	3	3	3	3	400	340	0	0	2	2	4	650	nil	nil	nil	nil	nil
56	C	SULAIMAN	40	MALE	324102	1	2	3	3	3	3	3	3	410	370	0	0	2	2	4	720	nil	nil	nil	nil	nil
57	C	GANDHIMATHI	50	FEMALE	37142	1	2	2	3	4	4	4	4	380	376	0	0	2	2	4	800	nil	nil	nil	nil	nil
58	C	MANIAMMAL	46	FEMALE	39140	2	2	2	3	3	4	4	4	360	380	0	0	0	2	4	840	nil	nil	nil	nil	nil
59	C	MUTHIAH	49	MALE	30119	1	2	2	3	3	3	3	3	420	370	0	0	2	2	4	680	nil	nil	nil	nil	nil
60	C	PALAMMAL	54	FEMALE	39141		2	2	2	3	3	3	3	390	360	0	0	0	2	4	780	nil	nil	nil	nil	nil